VOLUME 9 NUMBER 6

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

JUNE, 1923

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as second-class matter, Jan. 7, 1919, at the postoffice at Chicago, Illinois, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized Jan. 15, 1919.

CONTENTS OF PREVIOUS NUMBERS

APRIL, 1923. NUMBER 4

Teratoid Cyst of the Hypophysis. J. H. Globus, M.D., New York.

Charcot-Marie Atrophy: The Report of a Case with Necropsy. Charles S. Potts, M.D., and George Wilson, M.D., Philadelphia.

Recent Developments in Electrodiagnosis. Percival Bailey, M.D., Ph.D., Boston.

Arsphenamine Therapy in State Hospitals for Mental Disorders. George W. Mills, M.D., and Charles L. Vaux, M.D., Central Islip, N. Y.

Schizophrenic Catatonia with Associated Metabolic and Vegetative Features. Theophile Raphael, A.M., M.D., John Paul Parsons, B.S., M.D., and Margaret Newell Woodwell, A.B., Ann Arbor, Mich.

Phenobarbital Treatment of Insane Epileptics. Ira A. Darling, M.D., Warren, Pa.

The Pathogenesis of Epilepsy. Michael Osnato, M.D., New York.

Obituary: Robert Thaxter Edes, M.D.

News and Comment:
- Forty-Ninth Annual Meeting of the American Neurological Association.
- Annual Meeting of the American Psychiatric Association.

Abstracts from Current Literature.
- Spirochetes in the Central Nervous System in General Paralysis. — Parkinson's Disease Following Encephalitis.—Sequelae of Lethargic Encephalitis.—General Protein Therapy and Active Immunization of General Paralysis. — Congenital Megacolon Associated with Mental Disorder. — Severe Changes in the Nuclei of the Ganglion Cells in Dementia Praecox. — Frequent Minor Fits of Children.—The Female Castration Complex. — Treatment of General Paralysis by Inoculation with Malaria.—The Etiology of Phobias.—The "Nuquo-Mydriasis" Sign. — Cervical Neurofibroma. —Temperament in Harmonious Human Relationship. — Septum Lucidum Tumor. — Meningeal Symptoms of Ascariasis.—Plato: A Forerunner of Psychanalysis. — Vestibular Nerve Function in Myxedema. — Familial Infantile Bulbar Palsy.—Aortitis and Trigeminal Neuralgia.

Society Transactions:
- Boston Society of Psychiatry and Neurology.
- Chicago Neurological Society.
- Philadelphia Neurological Society.

Book Reviews.

MAY, 1923. NUMBER 5

Some Problems in Epilepsy. Walter Timme, M.D., New York.

The Ecology of Epilepsy:
- I. Epilepsy in Colonies. Charles L. Dana, M.D., New York.
- II. Racial and Geographic Distribution. C. B. Davenport, Ph.D., Cold Spring Harbor, N. Y.

Epilepsy and the Convulsive State. Foster Kennedy, M.D., New York.

Symptomatic Epilepsy in Birds. G. Mingazzini, M.D., Rome, Italy.

Infantile Progressive Muscular Atrophy of Werdnig-Hoffmann Type. Joseph C. Michael, M.D., Minneapolis.

Cystic Blastomycosis of the Cerebral Gray Matter Caused by Torula Hystolytica Stoddard and Cutter. Walter Freeman, M.D., and F. D. Weidman, M.D., Philadelphia.

Experimental Convulsions. Lewis J. Pollock, Chicago.

Studies in Epilepsy. Convulsions Experimentally Produced in Animals Compared with Convulsive States in Man. Charles A. Elsberg, M.D., and Byron P. Stookey, M.D., New York.

Metabolic States Contributing to Uremia. Nellis B. Foster, M.D., New York.

News and Comment:
- The Status of Epidemic Encephalitis.
- The Test of Hyperthyroidism.
- A Correction.
- Annual Meetings.

Abstracts from Current Literature:
- Antiproteolytic Ferments in Psychoses; Metabolism in Mental Disease.—Histopathologic Studies in Psychiatry.—Tuberculosis, Neuropsychopathy and Delinquency.—Hysterical Gangrene.—The Benzoin Colloidal Reaction.—Localization of Pain Sense in the Peritoneum. — Peruvian Folklore.—Sugar in the Blood and Spinal Fluid.—Spinal Actinomycosis.—Occipital Lobe Embolism.—Silent Intracranial Abscess.—The Influence of Pregnancy on the Incidence of Neurosyphilis.—Is the Stomach a Focus of Infection? — Treatment of General Paralysis with Relapsing Fever Virus.—The Viscosity of the Spinal Fluid.—Morphin Poisoning.—Botulism.—Blood Creatinin in Corpus Striatum Disorder.

Society Transactions:
- New York Neurological Society.
- Philadelphia Neurological Society.
- Chicago Neurological Society.

Book Reviews.

Archives of Neurology and Psychiatry

Vol. 9 JUNE, 1923 No. 6

THE MECHANISM AND THE FUNDAMENTAL CAUSE OF THE EPILEPSIES

JOSHUA ROSETT, M.D.
Instructor in Neurology, Columbia University
NEW YORK

CONTENTS

I. Introduction

II. Anatomic and Physiologic Considerations
- The State of Tonus and Its Dependence on the Suspension of Certain Cerebral Activities.
- The Biologic Significance of Posture. The Normal Periods of Suspended Cerebral Activity.
- Movement (clonic muscular contractions) as a Mode of Redistribution of Tonus.
- The Relation of Muscular Activity to the Conscious State.

III. The Normal Epileptoid Reaction
- The Epileptoid Reaction in Response to External Stimuli.
  1. The Stage of Disintegration of the Conscious State. The Aura.
  2. The Stage of Tonic Rigidity. The Paroxysmal Outcry. Anesthesia Associated with the Paroxysm.
  3. Clonic Movements.
  4. The Stage of Recovery.
- The Biologic and Physiologic Significance of the Normal Epileptoid Reaction.
- The Epileptoid Reaction Associated with Certain Functions.

IV. The Periodicity of the Normal Epileptoid Reaction

V. Experiments on the Effect of Caffein, Bromids and Phenobarbital on the Epileptoid Reaction and the Epileptic Paroxysm
- Experiments on Reptiles.
- Subjective Experiments.
- The Effect of Caffein and Phenobarbital on the Epileptic Manifestations.

VI. The Reciprocity of Nerve Functions. The Identity of the Epileptic Paroxysm with the Normal Epileptoid Reaction

VII. Summary

I. INTRODUCTION

The history of epileptology for the last hundred years is a record of sublime obstinacy in the face of increasing difficulties. With every addition of facts bearing on the disease, the hope of fathoming its etiology appeared more distant. In 1857 Brown-Séquard [1] was certain that he could produce epilepsy in animals by asphyxia or by injuries to the spinal cord. Sixty years later Silberstein,[2] having produced epileptiform attacks in animals by injuries to the brain and other portions of the nervous system, took pains to warn his readers that such injuries are not essential to the disease of epilepsy.

The production of movements simulating a localized convulsion by means of electrical or other excitation of certain areas of the cerebral cortex, has resulted in a wider knowledge of the anatomy and physiology of the central nervous system. Regarding epilepsy, however, the procedure has yielded little more information than the movements produced by stimulation of the spinal nerves. The application to the cortex, or the injection into the blood, of certain drugs, has taught the lesson that widely dissimilar substances, such as lead, the toxins of uremia, absinthe, or picrotoxin may, through their action on the central nervous system, produce convulsive movements. Yet this valuable lesson has furnished hardly any assistance to the understanding of the disease.

"If on the one side," wrote Reynolds [3] some sixty years ago, "it is said that convulsions are induced by cerebral anemia; by eccentric irritation; by injury to the nervous centers inflicted by disease on the brain and on the spinal cord, or experimentally produced by lesions of the former or the latter; by blood disease and by general cachexia and debility, it must be said in reply that epilepsy exists when no one of these conditions is present; and the utility of their observation, *quoad* the pathology of epilepsy, is to show how, when they are not present, the special phenomena of that disease may be brought about; and how when they are present they are related to the central fact of epilepsy."

It must indeed be plain that if any disease exists both in the presence and in the absence of a certain condition, that condition, much as it may contribute to increase or to diminish its severity, cannot be considered as the causative factor of the disease; and every condition in connection with epilepsy thus far observed has been open to this objection. The very multiplicity and variety of factors lately described by

1. Brown-Séquard, E.: Researches on Epilepsy, 1857.

2. Silberstein, A.: Experimentelle Untersuchungen über traumatische Epilepsie der Meerschweinchen, J. f. Psychiat. u. Neurol. **22**:123, 1916.

3. Reynolds, J. R.: Epilepsy, 1861, p. 50.

Kraepelin [4] as concomitant with different cases of epilepsy, is sufficient to convince anybody that such factors cannot but serve as contributories to the causation of the affection. Yet even if a constant factor concomitant with the symptom-complex of epilepsy were to be discovered, say of the nature of a chemical substance in the blood, proof would still be wanting that that factor is the cause of the disorder. Even if the application of such a substance to the cortex or its introduction into the blood of an animal regularly brought about a convulsion, we might still be justfied in doubting its responsibility for the disease. For we know, on the one hand, that chemical substances exist in the normal animal body whose application to the cortex produces serious alterations of its vascular supply. An example of such a substance is epinephrin. On the other hand, we know that the effect of the introduction into the body of foreign substances is no measure of the effects of those substances on the organism to which they are native.

In certain diseases of known etiology, which are characterized by periodicity, we can discover times of definitely increased activity of the causative factor corresponding to the periodic manifestations of the disease. Malaria is an example of such a disease. In epilepsy on the contrary, the causative factors thus far assumed are continuous in their action, while the disease is definitely, though irregularly, periodic.

A review of the literature on the subject of epilepsy reveals a singular fact. In the eager search of a great number of investigators for isolated phenomena analogous to the several manifestations of the epileptic fit—in the search for examples of hallucinations, of reduced states of consciousness, of tonic and clonic spasms—the examples of such manifestations nearest at hand have been overlooked. It will be seen in the course of the present investigation that the entire train of these manifestations, though in less striking form, nevertheless in the same sequence and with the same periodicity as in epileptics, occurs also in all admittedly nonepileptic persons possessed of the average mental and physical states of health. It will appear, moreover, that this train of manifestations subserves a necessary biologic function in itself, and also that it is an indispensable part of a number of other physiologic activities of the animal organism.

A study of epilepsy from this standpoint has thrown light on a number of hitherto obscure facts.

The beneficial effect of such drugs as the bromids and phenobarbital in diminishing the frequency and the severity of the epileptic paroxysm, is an established fact. These drugs are nerve depressants and hypnotics. Their action in reducing the state of consciousness is

4. Kraepelin, Emil: Zur Epilepsiefrage, Zeitschr. f. d. ges. Neurol. u. Psychiat. **52**:107, 1919.

well known. That the administration of drugs whose action is to reduce the state of consciousness should result in a diminution of the number and the severity of paroxysms, whose chief and frequently only characteristic is a reduction of the conscious state, appears to be a contradiction to the evidence of common experience. One of the results of the present investigation has been to reconcile this seeming contradiction.

Almost every investigator who has studied the disease, has found it necessary to call attention to an outstanding feature of its hereditary phases. An epileptic child may be born of nonepileptic antecedents. In such cases, however, it is generally found that the antecedents have been the subjects of neuropathy or psychopathy of one kind or another. Every investigator, too, has called attention to the fact that emotional shocks, infectious diseases, or traumatic injuries of any kind may produce the disease in a person whose nervous system deviates, however slightly, from the normal. The following two cases, from the series studied in the Neurological Division of the Vanderbilt Clinic, are illustrative of this point.

Case 1.—Joseph C., aged 20, with a history of normal birth, infancy and childhood, experienced at 13 a severe illness of a few days' duration that was diagnosed as sunstroke. A week or two after recovery there occurred a major epileptic paroxysm. Other attacks of both major and minor epilepsy followed with increasing frequency and there was progressive mental deterioration. He has forgotten a large portion of what he learned at school before the age of 13, and he has acquired no new knowledge since. No history of epilepsy or insanity could be traced in the family. The mother had been a very "nervous" woman as long as she could remember.

Examination showed the reflexes to be normal. Speech was slow and sentences were poorly formed. He stammered occasionally. His ears were abnormally large and stood out prominently from the side of his head. The ring finger of the right hand was small, thin, and connected with a web to the middle finger. The space between the second and third toes was webbed.

Case 2.—Charles S., aged 9, was reported to have had an easy birth without instrumental interference, though the head was abnormally large in proportion to the body. He was weak and sickly all through infancy. The first teeth appeared at 6 months. The first words were uttered at 2 years of age and he began to walk at the age of 4. At the age of 2 he had a severe febrile illness which began with a stiff neck and was of eight weeks' duration. At the age of 5 he had an attack of influenza that lasted a month. A week after recovery there was a paroxysm of major epilepsy, which has been repeated since on an average of once a week. No history of mental or physical defect in the family was elicited.

Examination showed the right side to be somewhat the weaker of the two and its patellar reflex slightly more pronounced. A positive Babinski sign was present on the same side. The abdominal reflexes were brisk. No abnormalities of the cranial nerves were found. There was no defect of coordination or equilibrium. The child's head was abnormally large. The left testis was undescended. Except for a rather precocious—a mannish—manner of conversation, there was no mental abnormality.

The congenital defect of the nervous system in the first case is inferred, though rather distantly, from the congenital physical defect of the webbed fingers and toes, and from a neuropathic antecedent. In the second, however, a congenital hydrocephalus bears a more proximate relation to a congenital abnormality of the nervous system. What bearing the undescended testis may have in this connection, we do not know. Whether the sign of Babinski, the exaggerated patellar reflex and the relative weakness of the right side date back to a hemiplegia at birth or to a meningitis at the age of 2, or to influenza at the age of 5, was not ascertained.

Both the congenital and the determining factors in the two cases are of widely different nature. They are examples of classes of cases to which every student of the disease has called attention. That different kinds of congenital defects, as well as different apparent determinants, should cause the appearance of the single symptom-complex of epilepsy is a point which has not been explained. It will appear from the present investigation that all such defects and determinants contribute to the enhancement of a normal reaction of a certain primitive order, to the extent of degrading it to the clinical picture of epilepsy.

Among the seemingly contradictory phenomena related to epilepsy is the fact that the same kind of stimulus is capable either of precipitating or aborting a paroxysm. Brown-Séquard [1] precipitated convulsive fits in his animals by peripheral irritation. Buzzard [5] found that the irritation of a blister will prevent an epileptic attack. The constriction by a band of an extremity in which the convulsion begins is known to abort an oncoming seizure. Gowers [6] and others speak of painful experiences as potent factors in precipitating a paroxysm. A loud noise, a whiff of ammonia, voluntary movements, and any number of most different conditions have been described as efficient in aborting a beginning attack; and it is the experience of every one who has made a study of the disease, that the same agencies are equally adequate in precipitating the paroxysms. Gowers,[6] indeed, describes the case of a patient in whom attacks were first precipitated by certain involuntary movements and, later, by voluntary movements as well. From the viewpoint of the present study the seemingly inconstant and contradictory effects on the epileptic of these different stimuli becomes easily explicable.

Among the factors which mark the appearance of the first paroxysm, that of fright occupies a place of great prominence. "Of the three forms of emotion," writes Gowers,[6] "fright takes the first place. . . .

5. Buzzard, Thomas: Diseases of the Nervous System, 1882, p. 425.

6. Gowers, Sir William R.: Epilepsy and Other Chronic Convulsive Diseases, 1901, pp. 25, 30 and 109.

The female sex is notoriously the more emotional, and accordingly the disease results from fright in a larger proportion of females than of males. . . . In childhood one sex is almost as emotional as the other, but with puberty men become far less emotional than women. The influence of fright as a cause of epilepsy is in strict harmony with this fact." Throughout the literature on the subject no reason for so close a connection between fright and the epileptic paroxysm is to be found, and the rather vague assumption is everywhere made that strong emotions have the general effect of increasing the susceptibility to epilepsy of persons of a neuropathic disposition. A much more exact reason for the close relationship between fright and epilepsy has appeared in the course of the present study.

The subnormal mental and moral traits which characterize a number of epileptics have given rise to much conjecture, more mystification, and not a little of mystic and phantastic interpretation. They have been studied and described with an admirable degree of insight and thoroughness by Echeverria,[7] Arndt,[8] Binswanger,[9] Spratling,[10] Régis,[11] Clark,[12] and a number of others. The present investigation corroborates the findings of these authors. The reason assigned for the peculiarity of character on the basis of the present study, will be found, at its conclusion, to be comparatively simple and understandable.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

While it is generally admitted that the epileptic person, unconscious and rigid, or writhing in convulsions, is in a state similar to that of the decerebrate animal, writers on epilepsy continue, with a singular inconsistency, to speak of the epileptic motor phenomena in terms of cerebral cortical discharges. The obvious reason for this inconsistency is an effort to align two known facts with the epileptic convulsion. One fact has reference to the twitching movements produced by electrical or other excitation of the motor area of the cerebral cortex. The other is the fact that lesions of limited areas in or near the motor cortex are productive of localized or Jacksonian convulsions, the musculature involved corresponding to the area of the cortex which is known to preside over their contractions.

It will be shown later that the twitching movements produced by the experimental excitation of the cortex are most probably due to a

7. Echeverria, M. G.: On Epilepsy, 1870, p. 362.
8. Arndt, R.: Lehrbuch der Psychiatrie, 1883, p. 405.
9. Binswanger, Otto: Die Epilepsie, Nothnagel's System of Medicine, **12**:313.
10. Spratling, W. P.: Epilepsy, 1904, pp. 287 and 438.
11. Régis, E.: Précis de psychiatrie, 1909, p. 881.
12. Clark, L. P.: Clinical Studies in Epilepsy, 1917.

release of motor function in certain groups of cells in the ventral portion of the spinal cord. The same principle holds good respecting the localized convulsions caused by localized lesions of the motor cortex. Far from being brought about by cortical "discharges," the results of the last twenty years' work in nerve physiology point indeed definitely to the conclusion that these convulsions are due rather to *the lack* of such discharges.

In order to rectify such misunderstandings as well as to afford a clearer view of the manner in which the epileptic attack originates in the particular biologic reaction disclosed by the present investigation, it becomes necessary to review, even though briefly, the facts relating to some of the nerve structures and functions most probably involved.

THE STATE OF TONUS AND ITS DEPENDENCE ON THE SUSPENSION OF CERTAIN CEREBRAL ACTIVITIES

Clinicians noted many years ago that among the results of lesions which destroy a large portion of the cerebrum is a state of general muscular rigidity, usually manifested by tonic fits. Such a case was reported by Bastian [13] in 1884. In the course of the next twenty years the increasing attention bestowed on this state of rigidity is evidenced by the greater detail of the descriptions. Thus, in the report of a case of intracranial aneurysm in 1904, Bruce and Drummond [14] note that during the spasms "the limbs became rigid, the arms were extended in front of the body and rotated so that the hands were placed back to back." In the same year, Stewart and Holmes,[15] in their treatise on the symptomatology of cerebellar disease, described cases exhibiting numerous phases of these abnormal muscular conditions with a degree of clearness that enabled the clinician to recognize them in previously obscure instances. Thus, in connection with a case of tumor of the under surface of the vermis and subsequent operative injury to the dentate nucleus, these authors describe a series of tonic fits in which "both arms were rigid and resistant to passive movement. . . They were adducted to the sides, extended at the elbows and wrists, while the fingers were flexed at all the joints and covered the thumb."

While clinicians continued to study such cases with increasing attention, experimental physiology was locating the center of muscular rigidity and elucidating its mechanism. The striking phenomenon of extensor rigidity of the decerebrate animal demanded an explanation,

13. Bastian, H. C.: A Case of Apoplexy in a Boy Aged 15, Trans. Clin. Soc. **17**:21, 1884.

14. Bruce, Alexander, and Drummond, W. B.: A Case of Intracranial Aneurism in a Young Subject, Rev. Neurol. & Psychiat. **2**:737, 1904.

15. Stewart, T. G., and Holmes, Gordon: Symptomatology of Cerebellar Tumours, Brain, **27**:522, 1904.

and efforts were made to discover the pathways which, in the normal state of the animal, maintain its musculature in a moderate state of tonus. In 1897, Löwenthal and Horsley,[16] gave the result of their observations on the relation of the cerebellar to the cerebral structures as follows: "When both cerebral hemispheres were removed and, as a result, active extension tonus of the limbs was obtained, excitation (faradic) of the upper surface of the cerebellum caused immediate relaxation of such tonus so long as the current was applied."

In 1902, Fröhlich and Sherrington [17] attempted to trace the origin of the impulses which give rise to and which abolish the tonic state of the musculature in the decerebrate animal. They found that electrical excitation of the direct cerebellar tract in the spinal cord increased the rigidity of the homonymous limb, and that there existed a pathway in the ventrolateral column of the spinal cord, excitation of which resulted in the inhibition of rigidity in certain muscles. They concluded that "the path of inhibition in the ventrolateral white column may be connected with the afferent nerves from the crossed hind limb." Later, Sherrington [18] found that one of the prime conditions necessary for the production of the tonic state in the decerebrate animal consisted in afferent impulses arising in the muscles themselves. "The de-afferented preparation," he writes, "exhibited no trace of decerebrate rigidity, the 'normal' preparation always did." Still later the same investigator [19] proved that the decerebrate attitude was identical with the reflex act of standing; "That this is so may be demonstrated by setting the decerebrate animal on its feet; it is then seen that the preparation stands."

The experiments of Graham Brown [20] proved beyond a doubt that the center responsible for the tonic state of the decerebrate animal is the nucleus ruber of the midbrain. Section of the midbrain in front of the anterior colliculi resulted in extensor rigidity. Section below the posterior colliculi abolished the rigidity. Stimulation of various points in the cross section of the midbrain resulted in the assumption of definite postures of the body and limbs of the animal. These postures, moreover, continued to be maintained after the cessation of the

16. Löwenthal, M., and Horsley, Victor: On the Relation Between the Cerebellar and Other Centres (namely, Cerebral and Spinal), with Especial Reference to the Action of Antagonistic Muscles, Proc. Roy. Soc. **61**:20, 1897.

17. Fröhlich, A., and Sherrington, C. S.: Path of Impulses for Inhibition Under Decerebrate Rigidity, J. Physiol. **28**:14, 1902.

18. Sherrington, C. S.: On Plastic Tonus and the Proprioceptive Reflexes, Quart. J. Exper. Physiol. **2**:109, 1909.

19. Sherrington, C. S.: Postural Activity of Muscle and Nerve, Brain **38**: 191, 1915.

20. Brown, T. Graham: On Postural and Nonpostural Activities of the Midbrain, Proc. Roy. Soc. **87**:145, 1913.

action of the stimulus. "The area includes that of the nucleus ruber and of the posterior longitudinal bundle. . . . Stimulation within this area . . . is accompanied by the assumption of a definite posture on the part of the animal," and again[21]: "The main effect of stimulation of one red nucleus is a movement of flexion of the arm of the same side and a movement of extension in the opposite arm . . . those movements having attained their maximum, tending to be maintained long after cessation of the evocative stimulus."

The center for tonus having been thus located, the important question remained: What are the cerebral pathways which, in the normal state, keep the tonic function of the red nucleus in abeyance? The question was answered by Weed,[22] who traced that pathway through the internal capsule, the crus cerebri, the middle cerebellar peduncle and the vermis of the cerebellum. That system is the frontopontocerebellorubral pathway. Electrical stimulation of this pathway resulted in an abatement of the decerebrate rigidity.

The conclusions of Weed were fully corroborated by later investigations. Thus, for example, Cobb and others,[23] give as one of the results of their investigations the following "Stimulation of the superior cerebellar arm in decerebrate cats causes ipsilateral inhibition of their extensor rigidity."

The state of tonus, then, depends on two sets of opposite impulses. One, the proprioceptive, arising in the muscles, passes along the spinocerebellar tracts, and by way of the cerebellum, arrives at the red nucleus. The interruption of this pathway does away with the tonic activity of the red nucleus. The other set of impulses is of a kind which, in the presence of afferent stimuli, maintains the tonic activity of the red nucleus in abeyance. It takes its origin in the cerebral cortex and, by way of the cerebropontine system of fibers, passes into the cerebellum and so arrives at the red nucleus of the midbrain.

The cerebellum thus becomes the meeting place of two sets of impulses of opposite function. The uncertain results on the tonic function brought about by gross injuries to the cerebellum are easily accounted for by the fact that the fibers of the two pathways are, within that organ, very much intermingled, though more so in some situations than in others. This may be judged in part from the figure of the author's dissection (Fig. 1).

---

21. Brown, T.: On the Effect of Artificial Stimulation of the Red Nucleus in the Anthropoid Ape, J. Physiol. **49**:185, 1915.

22. Weed, L. H.: Observations on Decerebrate Rigidity, J. Physiol. **48**:205, 1914.

23. Cobb, S., Bailey, A. A., and Holtz, P. R.: Genesis and Inhibition of Extensor Rigidity, Am. J. Physiol. **44**:239, 1917.

A fact of great importance in connection with the state of rigidity is that the posture of the animal as a whole is determined by the position of certain of its parts. Thus Magnus and de Kleijn [24] showed that the position of the body of the decerebrate animal is changed by the passive rotation of its head. The removal of the

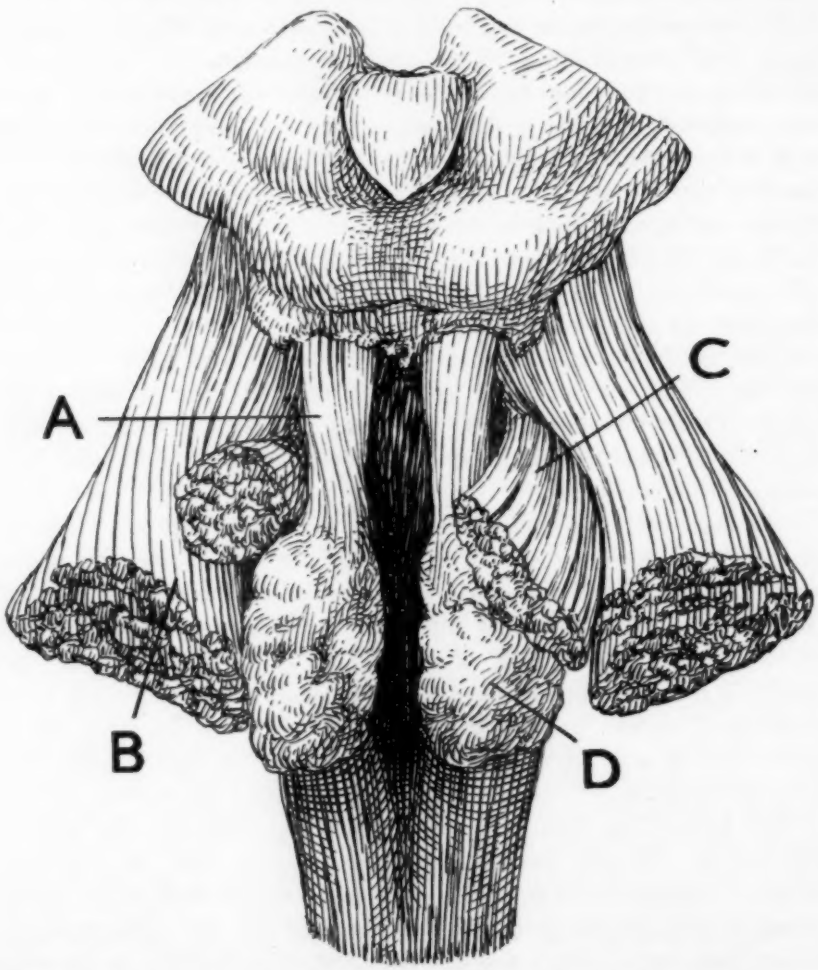


Fig. 1.—A dissection showing the relations of the peduncles to each other. *A*, superior peduncle; *B*, middle peduncle; *C*, inferior peduncle; *D*, dentate nucleus.

so-called neopallium alone with subsequent extirpation of one labyrinth results in a definite posture of decerebrate rigidity. Barenne and

24. Kleijn, A. de, and Magnus, R.: Beiträge zum Problem der Körperstellung, Pflüger's Arch. **180**:291, 1920.

Magnus [25] showed that when the head of such an animal is passively rotated, its body and limbs assume a different posture. Jonkhoff [26] describes a case of status epilepticus which illustrates the dependence of the posture of some of the muscles on that of others. The patient was unconscious and rigid. When the head, which was turned to the right, was passively rotated to the left, the extension of the right arm disappeared. These are examples of the manner in which the afferent impulses arriving from the muscles play on the center of tonus and, by accentuating the activity of certain of its parts, bring about a movement which results in a change of posture.

The vestibular mechanism, which plays a very important rôle in the maintenance of certain postures of the body, does not concern us in this connection.

The foregoing physiological facts have been turned to account by Wilson,[27] who has shown that a number of diseases in man associated with tonic rigidity or tonic spasms have been found at necropsy to be cases of practical decerebration, complete or partial. He concludes that: "Any lesion dissociating the cortex from the mesencephalocerebellar level and at the same time leaving the latter functionally intact is calculated to cause the rigidity to develop." In order to accentuate the importance of the inhibitory action of the cerebrum on the red nucleus, this investigator then adds that: "there is no question that the phenomena of decerebrate rigidity are release phenomena."

We may thus conclude that the inhibitory pathway in question comprises the entire cerebropontine radiation.

In order to obviate possible controversy, it is perhaps as well at the outset to throw one question out of court, namely, whether the action of a certain nerve impulse in extinguishing that of another is in the nature of an active inhibition or whether it is only apparently so. "A natural surmise," says Sherrington,[28] "seems that it may be in the nature of interference, somewhat as waves of sound or light by interference mutually extinguish each other." Bayliss [29] indeed explains this phenomenon on the physical basis of the extinction of a wave of force when two superimposed waves are so timed as to alternate in half phases; in other words, when the hollows of one are filled in by

25. Barrenne, J. G. Dusser de, and Magnus, R.: Beiträge zum Problem der Körperstellung, Pflüger's Archiv. **180**:75, 1920.

26. Jonkhoff, D. J.: The Prognostic Importance of the Magnus de Kleijn Reflexes in Man, Nederl. Tijdschr. Geneesk. **64**:307, 1920; abstracted in J. Nerv. & Ment. Dis. **55**:519, 1922.

27. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, Brain **43**:220, 1920.

28. Sherrington, C. S.: Reflex Inhibition as a Factor in the Coordination of Movements and Postures, Quart. J. Exper. Physiol. **6**:251, 1913.

29. Bayliss, W. M.: Principles of General Physiology, 1915, p. 420.

the protuberances of the other. Keith Lucas [30] has shown such an interference of one nerve impulse with that of another to be equivalent to an impulse passing through a region of increasing decrement. In the course of a study of the simplest reflex arc, Veszi [31] was able to demonstrate the fact that a muscular contraction induced by the excitation of one posterior nerve root may be entirely inhibited by the excitation of another posterior root. Tilney [32] offers the simple explanation of the complete neutralization of one force by another in a parallelogram in which forces of the same magnitude work in opposite directions.

The important fact bearing on the subject with which we are concerned is that the moderation of the activity of the red nucleus, in the normal state of the adult, depends on certain cortical activities propagated to the midbrain; and that when these activities are suspended, the nucleus of the midbrain, no longer impeded in the exercise of its function, brings about a state of general muscular rigidity. The exact nature of the cortical activities which impede the function of tonus of the red nucleus is quite immaterial from the standpoint of the present study.

Although the midbrain occupies a position of preeminence in the function of tonus, its monopoly over this function is not complete. The musculature of the dog or cat whose spinal cord has been transected.[18] recovers after a time a certain amount of its former tonicity. Pike [33] attributes this partial recovery to structures in the cord which remain dormant while the more highly evolved mechanisms can perform the same function in a more efficient manner, but which become active when these are removed. It is undoubtedly true that during the course of evolution of the nervous system, the transference of certain functions from lower to higher levels has gone far in advance of the transference of the structures with which those functions are associated. For while we know that the course of organic evolution is characterized not only by aggregation but by a simultaneous segregation of structures and functions as well, the latter process is practically never complete.

THE BIOLOGIC SIGNIFICANCE OF POSTURE. THE NORMAL PERIODS OF SUSPENDED CEREBRAL ACTIVITY

The special importance of posture arises from the fact that animals are made up of jointed segments. With respect to the decerebrate

30. Lucas, Keith: The Conduction of the Nervous Impulse, 1917, p. 93.

31. Veszi, J.: Der einfachste Reflexbogen im Rückenmark, Zeitschr. f. allgm. Physiol. **11**:168, 1910.

32. Tilney, Frederick: A personal communication.

33. Pike, F. H.: Studies in the Physiology of the Nervous System, Am. J. Physiol. **24**:124, 1909.

animal it is undoubtedly true that[19] "the distribution of this reflex tonicity embraces just those muscles whose contraction tends in the erect position of the animal to counteract the effect of gravity." With respect to the intact animal, however, the function of tonus has another equally important use. In order to enable any distal segment of the body or the limbs to execute a measured movement, the central segment to which it is jointed, must be fixed. Thus in order that the forearm may move for a definite distance in a definite direction, the arm must be fixed at the shoulder. Fixation of the shoulder, complete or partial, according to the necessity of the occasion, is accomplished by a tonic contraction of the muscles which pass over the joint on opposite sides. And it is this fixation which constitutes the special posture of the upper limb in certain movements of the forearm.

In the ordinary activities of the animal, occasions must be very numerous when joints are partly or wholly immobilized for varying durations of time by the contraction of the overlying musculature, in order to afford a steady base for the movement of relatively distal segments of the body and limbs. We have seen that the special nerve center which presides over the production of this fixation of the joints —over the maintenance of posture—is the red nucleus. We have further seen that the activity of that nucleus, in the presence of certain stimuli, is dependent on the extinction of certain impulses arriving from the cerebral mantle. Every possible movement of the animal, implying as such movement does the temporary tonic contraction of certain groups of muscles, must also imply a simultaneous temporary extinction of certain cerebral activities. When considering the epileptic paroxysm as it occurs in the severer form in the epileptic, and in the milder form in all normal persons, we shall have an opportunity for a more concrete application of the foregoing facts.

MOVEMENT (CLONIC MUSCULAR CONTRACTIONS) AS A MODE OF REDISTRIBUTION OF TONUS

In order to explain the phenomenon of movement, as distinguished from that of posture, certain investigators have found it necessary to postulate the existence of special anatomic structures, outside of those devoted to the function of tonus and the inhibition of tonus.

"While a state of momentary decerebration," writes Sargent,[34] "may be held to explain the tonic stage of the epileptic fit it is not easy to account for the clonic movements. Kinnier Wilson . . . having characterized the tonic fits he observed in the cases which he was describing as 'attacks of decerebration,' remarks that the absence of any clonic movement is of much significance. He takes it to indicate that clonic movements have different origins and are the expression of

34. Sargent, P. W. G.: Some Observations on Epilepsy, Brain **44**:312, 1921.

the activity of different motor mechanisms, the one being characterized by static, the other by phasic activity."

J. Ramsey Hunt [35] postulates "two distinct components, each represented throughout the entire efferent nervous system by separate neural mechanisms which are physiologically and anatomically distinct. One is the movement proper, which is subserved by the *kinetic system* (motion system). The other represents that more passive form of contractility which we recognize in tonus, posture and equilibrium, and is subserved by the *static system* (posture system)."

In the light of the known anatomic and physiologic facts, the assumption of special systems devoted to *movement as a distinct physiologic entity,* appears to be quite gratuitous, as may be gathered from the following considerations:

Since the common final motor pathway has its origin in the ventral gray columns of the spinal cord and brain stem nuclei, it must be plain that the probable action of the red nucleus, however indirectly, is on the motor cells of the cord. Wilson [27] indeed concludes that "we cannot believe that in the red nucleus we have other than a re-presentation of the ventral cord cell groups," and that "stimulation of a point in the red nucleus . . . may be supposed to innervate a physiologic group of ventral cells."

It is a fact that injury to the cerebrospinal tract in any part of its course results in a state of increased tonus of the musculature below the level of the lesion.

If the tonic action of the musculature is to be ascribed to the influence of the red nucleus on the motor cells of the spinal cord, then the increased tonus resulting from pyramidal lesions must be attributed to removal of an inhibitory influence which that tract normally exerts on the ventral motor cells. In this respect, therefore, the cerebrospinal tract must exercise an influence on a lower level similar to that which the cerebropontine tract exerts on a higher level. Such an assumption, it will be presently seen, is not in the least incompatible with the fact that stimulation of the area of origin of the cerebrospinal pathway—the precentral—results in movement.

The influence of the pyramidal fibers on the ventral cells of the cord is most probably by means of an intermediate neuron. Each of the pyramidal fibers is thus unabled to transmit an impulse to a number of ventral cells. We know, moreover, that the groups of cells thus innervated by the pyramidal fibers correspond to definite groups of muscles. The simplest volitional movement, say that of flexion at the elbow, involves the synergic contraction of a number of flexor muscles with a simultaneous inhibition of a number of extensors. The details of

35. Hunt, J. R.: Dyssynergia Cerebellaris Myoclonica, Brain **44**:490, 1921.

the mechanism of reciprocal innervation of antagonistic muscles which Sherrington [36] called "co-operative co-ordination" cannot but be found in a patterned arrangement of groups of motor cells in the spinal cord.

The particular force which brings about muscular contraction is, in our ignorance of its ultimate nature, known as muscle tonus. When the amount of tonus of one set of muscles is exactly counterbalanced by an equivalent amount of tonus of an antagonistic set, the limb remains at a standstill, in a definite posture. Any difference in the amount of tonus of the two respective sets of muscles—any difference of potential—manifests itself in movement; and the movement continues until a balance between the forces is again established. At that moment the limb is once more in a definite posture.

We are thus enabled to understand why electrical excitation of the precentral area is productive of movement. A stimulus passing along the pyramidal fibers to the cells which innervate, say, a group of extensors, must cause an abatement of the extensor tonus. The consequent predominance of flexor tonus will then result in a movement of flexion. Once the contraction of flexors has begun, it will proceed with an increment of force for the following reason:

The tonic action of the nucleus of the midbrain depends on certain afferent impulses. These impulses have been shown by Sherrington [18] to originate in the muscles themselves. The condition for their origin is the approximation of the points of insertion of the muscles. The beginning contraction of a muscle, therefore, by propelling stimuli to the midbrain, increases the tonic activity of the red nucleus and receives from it in turn increments of force.

Again, we know that stimulation of various points in the tonic mechanism results in the assumption of definite postures. Stimulation of different degrees of intensity and of different points in that mechanism must result, therefore, in *changes* of posture; and movement is nothing but a succession of such changes. These, we have seen, may result from alterations in the activity of the proprioceptive mechanism, as in the Magnus and de Kleijn reflexes. The rhythmic movements of progression of Weed's [37] decerebrate kittens exemplify the manner in which a succession of proprioceptive stimuli is capable of producing a succession of postures. And the same is true respecting the coordinate movements obtained by Graham Brown [38] in spinal and decerebrate preparations. Other factors are equally potent in producing changes of

36. Sherrington, C. S.: On Reciprocal Innervation of Antagonistic Muscles, Proc. Roy. Soc. **60**:414, 1897.

37. Weed, L. H.: The Reactions of Kittens After Decerebration, Am. J. Physiol. **43**:131, 1917.

38. Brown, T. G.: Studies in the Physiology of the Nervous System, Quart. J. Exper. Physiol. **6**:25, 1913.

posture, as may be gathered from the following:[28] "Take the case where a reflex (decerebrate) preparation is exhibiting the steady postural reflex of standing, and then draws up one foot on that foot being pinched. . . . In the transition from one reflex act to another, a muscle's activity is inhibited if it would offer obstruction to the new reflex."

Our conception of movement, then, is simply one of redistribution of tonus—the leap from one posture to another. Such a conception corresponds with the effect of the process on the environment. This effect is *work done,* which John Stuart Mill[39] defined as *a change in the position of objects.*

Such a conception of movement, too, enables us to understand how localized clonic contractions of muscles may be caused by localized injuries of the motor area of the cortex. When certain points in the tonic mechanism of the midbrain or of the spinal cord are released from the impeding action of certain portions of the cerebropontine or the cerebrospinal systems, their functional activity remains determined by afferent impulses only. The periodic ebb and flow of such afferent impulses must therefore result in periodic convulsions of the parts of the body whose musculature is presided over by corresponding patterns in the tonic mechanism.

THE RELATION OF MUSCULAR ACTIVITY TO THE CONSCIOUS STATE

Before proceeding with facts regarding the occurrence of the several forms of the epileptoid paroxysm in normal persons, the relation in which muscular contraction stands to the conscious state must be elucidated.

The terms consciousness and unconsciousness, as employed in the present study, are meant to convey but a general meaning. The former, as it is applied to the wide-awake state of the average normal person; the latter, as it might be applied to profoundly comatose conditions; and the innumerable degrees of transition between the two states, as these might exist in a person who passes from the normal waking state into one of profound coma.

That merely coordinated movement is not in itself a measure of the degree or scope of the conscious state, is evidenced by the movements executed in states of automatism, in delirium, as well as by the animal with an ablated cortex, or by the spinal frog. Even when such movements subserve both immediate and distant benefits, they are not necessarily a sign of the fully conscious state. Thus, a pedestrian, on

39. Mill, John Stuart: On the Requisites of Production. Principles of Political Economy, 1891.

his way to transact some business previously determined on may, while walking, be in that semiconscious state which is known as "absent-mindedness." Yet his act of walking, automatic as it is, may subserve both immediate and distant benefits.

All such examples of movement, no matter how simple or how complex are, however, marked by the common characteristic that at the time of their execution they are beyond the control of the individual. From the viewpoint of movement, therefore, it is the ability to control muscular contraction which is the measure of the degree of the conscious state at any one time.

Much less obvious than the preceding is the proposition that even though a muscular contraction be initiated voluntarily, the contraction itself implies a certain reduction in the conscious state which is in proportion to the intensity of the muscular activity. Without an appreciation of the facts involved in this proposition it is not possible to understand the connection between the reduced state of consciousness and the muscular phenomena either in the normal epileptoid or in the epileptic paroxysms. It is, therefore, important to deal with them in this place.

The fact that even very strong stimuli may be prevented from reaching the sphere of consciousness by voluntarily initiated muscular contractions, bears testimony to the truth of the foregoing proposition. Darwin [40] called attention to the incompatibility of the conscious state and that of muscular activity as follows: "A man cannot think deeply and exert his utmost muscular force. . . . Sailors who are going to be flogged sometimes take a piece of lead into their mouths in order to bite it with their utmost force and thus to bear the pain. Parturient women prepare to exert their muscles to the utmost in order to relieve their sufferings."

Whoever has suffered from an intense toothache will testify to the fact that the pain is much more severe when resting quietly in bed than when "pacing the floor." Persons afflicted with worry "pace the floor" and are generally "on the go." Such muscular exercise, by reducing in part the conscious state, offers them a degree of relief.

A probable explanation of such a relation of muscular contractions to varying states of consciousness may be gathered from the consideration of the production and distribution of tonus:

We have seen that one of the conditions for setting in action the tonic mechanism of the midbrain is a suspension of the function of the cerebropontine system. That system is the largest cerebral efferent fiber system. It descends from a number of points throughout the

40. Darwin, Charles: The Expression of the Emotions in Man and Animals, 1873, p. 72.

extent of the cerebral cortex, with the possible exception of the lowest portion of the temporal and occipital regions. Its extent may be judged in part from Figure 2 of the author's dissection. The abandonment of function by so widely distributed a system of neurons must result in profound dissociations of the neural mechanisms with which it is connected in the cerebral cortex; and such dissociation is equivalent to a reduction in the conscious state.

The truth of the foregoing considerations may be tested in several ways. If the reduction of the conscious state is a necessary condition for the production of muscular contractions, then any degree of narrowing the field of consciousness should result in some degree of muscular activity, and different degrees of contractions of the field of consciousness should result in corresponding degrees of muscular activity.

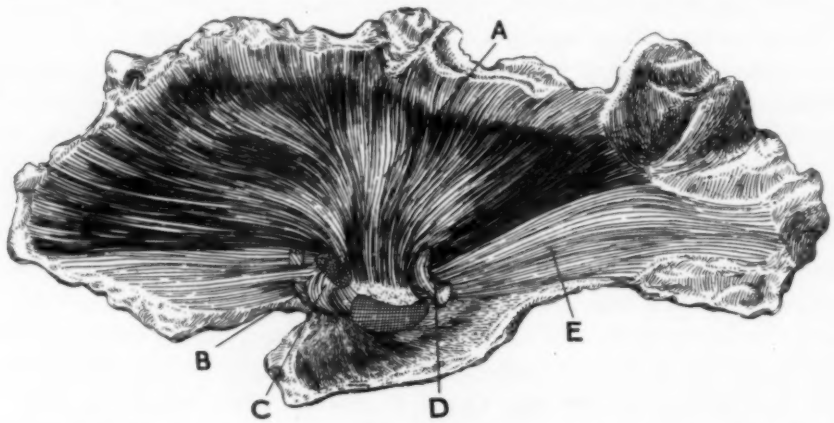


Fig. 2.—Mesial view of the cerebral structures exposed on removal of the thalamus and the thalamic fiber system. *A*, palliopontine and pyramidal systems; *B*, anterior commissure; *C*, pallidal fiber system *X*; *D*, internal geniculate body and auditory tract; *E*, an *X* fiber system converging on the globus pallidus.

The field, or scope of consciousness may be narrowed by the act of concentration of attention. That the latter act really implies a contraction of the field of consciousness may be easily gathered from the fact that the person whose attention is "absorbed" is to a great extent unconscious of his surroundings and that he may indeed be insensitive to a number of even very strong stimuli. It is a familiar fact that when such a person, by dint of repeated requests, is finally aroused to reply, he manifests a total ignorance of the previous attempts to arouse him.

The act of concentration of attention is accompanied by certain muscular activities. Lindley's [41] study of 622 pupils in the schoolroom

41. Lindley, E. H.: A Preliminary Study of Some of the Motor Phenomena of Mental Effort, Am. J. Psychol. **7**:491, 1895.

led him to the conclusion that varying degrees of mental concentration manifested themselves in certain sustained postures and automatic movements. Stein [42] describes clonic movements which she observed when attention wandered, that is to say, when it shifted from point to point. The following two experiments, performed by the author a number of times, may be repeated by anybody with little trouble and much profit.

The first consists in concentrating the attention on a single point or on the succession of a single sound, such as the falling of water drop by drop. It will then be found that as long as attention is sustained, a single posture of the body and the limbs is sustained as well; and the posture is involuntarily changed immediately attention begins to shift. The reverse is likewise true. Any attempt to change the fixed posture results in the shifting of the attention.

The second experiment consists in the solution of a problem involving sets of facts each of which requires separate careful consideration. It will be found that simultaneously with the shifting of the thought from one set of facts to another, there is an involuntary change of posture.

Féré [43] found that the concentration of a person's attention by sharp and penetrating odors increased his capacity for muscular exertion. The so-called psychogalvanic reflex has its origin, in part at least, in the muscular contractions which ensue when the scope of consciousness is reduced within certain limits by definite sensations, or emotions. By an improvement of Hamlet's [44] method, Peterson and Jung [45] obtained an electrical response when certain words which compelled the subject's special attention were read to him. The findings of these investigators, as well as the similar findings of Féré,[46] of Veraguth,[47] and others, were at first attributed to other causes than muscular activity. Sidis and Nelson,[48] however, in their experiments

42. Stein, G.: Cultivated Motor Automatism, Psychol. Rev. **5**:295, 1898.

43. Féré, C.: Les variations de l'excitabilité dans la fatigue, L'année psychol. **7**:69, 1900.

44. Hamlet. " . . . The play's the thing
Wherein I'll catch the conscience of the King.
. . . . . .
One scene of it comes near the circumstance
Which I have told thee of my father's death.
I prithee, when thou seest that act a-foot,
Even with the very comment of thy soul
Observe my uncle . . . "

45. Peterson, F., and Jung, C. G.: Psychophysical Investigations with the Galvanometer, Brain **30**:153, 1907.

46. Féré, C.: Note sur des modifications de la résistance electrique sous l'influence des excitations sensorielles et des émotions, Compt. rend. Soc. de Biol. **5**:217, 1888.

47. Veraguth, O.: Abstracted in Arch. d. Psychol. **6**:162, 1916.

48. Sidis, Boris, and Nelson, L.: The Nature and Causation of the Galvanic Phenomenon, Psychol. Rev. **17**:98, 1910.

on animals came to the conclusion that the electrical current was produced by muscular contraction. In summing up the results obtained by different investigators in this line of research, Prideaux [49] concludes that the so-called psychogalvanic reflex which is concomitant with the experience of certain emotions is, in part at least, due to muscular contraction. It is, therefore, in part, really what is now known as a muscle action current.

The relation between muscular contractions and the conscious state, therefore, may be likened to the two arms of a lever; the ascent of one arm involves the descent of the other. Each muscular contraction, implying as it does a release of muscle-tonus, implies at the same time a suspension of a certain amount of cerebral activity—a certain reduction in the conscious state.

III. THE NORMAL EPILEPTOID REACTION

The phenomena which constitute the normal epileptoid paroxysms, like those associated with true epilepsy, exhibit a wide variation in the intensity and duration of each attack as a whole, as well as in the separate manifestations. Thus, for example, that stage of the epileptic attack during which the disintegration of the patient's consciousness marks its progress on the way to extinction, may be of such long duration, that the dominant symptom of the attack is one of elaborate reactions to equally elaborate hallucinations. On the other hand, the transition from the wide-awake state to that of utter unconsciousness may be sufficiently rapid to escape the notice of the keenest observer. And the same is true of the other manifestations of the attack. Notwithstanding, however, the wide variation in the degree of intensity and duration of the separate manifestations of the epileptic paroxysm, the sequence in which they follow each other in the more pronounced cases is, on the whole, uniform. This sequence is as follows: (1) A preliminary stage, which consists of a reduction in the conscious state. (2) A state of muscular contraction, such that the tonus is equally distributed to antagonistic muscles, except for some predominance of certain extensors, pronators, and adductors. This equal distribution of tonus to antagonistic muscles results in immobilization of the joints and a consequent state of rigidity. When the rigidity is general and its progress rapid, its onset is marked by an expiratory sound, the epileptic cry. (3) A redistribution of the muscle-tonus, resulting in clonic movements. During this stage involuntary evacuation of the bladder or the rectum may take place. (4) A period of recovery, marked by exhaustion and a gradual integration of the previously disintegrated state of consciousness.

---

49. Prideaux, E.: The Psychogalvanic Reflex, Brain **43**:50, 1920.

These phenomena, in the same sequence in which they follow each other in the epileptic attack, occur also in all normal individuals under two sets of conditions: (1) on exposure to the wide range of circumstances which necessitate sudden movement, or the sudden concentration of attention; (2) in connection with the performance of certain functions. When considering the causes of the periodic recurrence of the epileptic paroxysm, it will be shown that the normal epileptoid paroxysm, no matter what its determining factors may be, is subject to the same law of periodicity.

THE EPILEPTOID REACTION IN RESPONSE TO EXTERNAL STIMULI

The general condition for the occurrence of the epileptoid reaction to be described under this heading is exposure to a stimulus so sudden as to take the person or the animal unawares.

The following case, although rather unusual, illustrates the severe form of the normal epileptoid paroxysm. The milder and the far more common forms which occur in the daily life of every person, will be pointed out in the consideration of the separate manifestations of the fit.

A young school teacher, James B., met with an accident on a farm in Woodstock, Ulster County, N. Y., in the fall of 1922. While picking apples, the branch on which he stood gave way. He fell from a height of some twelve feet and fractured three ribs. The young man was of light weight and agile, and the ground on which he fell was soft; it seemed therefore singular that he should have sustained so much injury. As the accident took place while the present study was in progress, the writer was especially interested, and the injured man as well as the members of his family, who were with him at the time of the accident, were questioned regarding the details of the occurrence. The following history was elicited:

The patient, healthy and intelligent, 23 years of age, stated that while picking apples in a tree, he suddenly heard the branch on which he stood crack. He next became aware of standing with a cup in his hand from which he was drinking water. It then occurred to him that something unusual must have happened, for he was surrounded by the members of his family who appeared to be solicitous about his condition. He remembered that he had been picking apples in the tree and that he had heard the branch break, and realized that he must have been unconscious for a time, for the spot where he stood with the cup in his hand was at a considerable distance from the fateful apple tree. He had no direct knowledge of the fall. Any such previous attack of unconsciousness was absolutely denied, and the nature of his occupation lends credence to this denial.

The members of his family, who were with him at the time of the accident, stated that James was picking apples in the tree when they heard him cry out and saw him fall to the ground. They ran to him and helped him rise. Although he appeared rather dazed and did not answer their questions, yet when led along towards the house, he appeared to walk in a fairly normal manner. After walking some distance, he asked for water, and when it was brought to him he drank it. A little later he complained of pain at the site of the injury.

The separate phenomena involved in this case will be considered in the succeeding sections.

1. *The Stage of Disintegration of the Conscious State. The Aura.*—The immediate effect of exposure to a sudden painful or emotional stimulus is a rapid contraction of the field of consciousness to within the limits occupied by the stimulus. It is the process known as "centering the attention." The extent and rapidity with which the field of consciousness is thus narrowed is dependent on the strength of the stimulus and the suddenness of its application on the one hand, and on the state of unpreparedness of the person on the other. In the experiments on the effect of suddenly applied stimuli made in connection with the present study the second stage, that of general rigidity, came on apparently without being ushered in by any reduction in the conscious state. In these experiments, however, the stimulus, though unexpectedly and rapidly applied, was necessarily very weak and the spasm of general rigidity, though very distinct, was at the same time exceedingly brief. It is to be expected, therefore, that the stage of unconsciousness in the case of exposure to the weaker kinds of emotional or painful stimuli, would largely escape observation. Cases of moderate fright, or of other emotional shocks, such as the hearing of a joyful or a distressing piece of news are, however, on record in which the initial reduction of the conscious state is very considerable. The accident related in the preceding section is a fair example of a class of cases in which the reduction of the conscious state is pronounced and the progress of the reduction rapid. When, however, such a reduction of consciousness is very gradual, it is a rule—and that rule holds good to a very large extent in epilepsy as well[50]—that it does not sink to the point of complete extinction. In such cases of gradual disintegration of consciousness, hallucinations and automatism make their appearance.

Auditory hallucinations occurring in states of "emotion" or "excitement" are mentioned by Schwartze,[51] Politzer,[52] and others. The former author describes these "physiological subjective auditory sensations" as occurring most frequently in the form of ringing sounds of different tone and loudness, appearing with great suddenness and vanishing gradually; more rarely these sounds consist of a number of tones which are repeated over and over again. One of the writer's

50. Gowers, Sir William R.: The Borderland of Epilepsy.

51. Schwartze, H.: Ueber subjective Gehörsempfindungen, Berlin. klin. Wchnschr., No. 12, p. 124, 1866.

52. Politzer, Adam: Ueber subjective Gehörsempfindungen, Wien. med. Wchnschr. No. 94, 1693, 1865.

patients, however, described his auditory sensations as resembling the noise of a bursting steam boiler with a subsequent hissing sound.

A number of the cases of sudden subjective blindness which have been conveniently stacked away under the heading of "hysterical amauroses" may be discovered, on a closer examination of the circumstances under which they occurred, to consist of hallucinatory experiences associated with the epileptoid reaction. The following case from Willbrand and Saenger [53] is an example:

> A seamstress, 20 years of age, had broken the needle while sewing on a machine, and a fragment flew off in the direction of her left eye. She immediately experienced a pain and lost vision completely in that eye. On examination the eye was found to be uninjured. Vision returned within three days.

Secular as well as religious literature is full of instances of visual hallucinations occurring in connection with states of surprise, anxiety and fear. The following from Job (iv, 14) is an example:

> "Fear came upon me and trembling, which made all my bones shake, Then a spirit passed before my face. . . . It stood still but I could not discern the form thereof: an image was before my eyes. . . . "

Cutaneous hallucinations—paresthesia—are exceedingly common even in the very mild cases in which the duration of the entire paroxysm is only a second or two. These are commonly described by the person as a sensation of numbness in the extremities or as that of cold running down the back. The "shiver running down the back" may be a muscular reaction to a cutaneous hallucination of central origin, or its origin may be local and induced by certain vasomotor changes associated with the attack. When the entire paroxysm is brief, these sensations outlast the tonic and clonic stages and persist during the stage of recovery.

Hallucinations of deep sensation may be easily inferred from such statements as: "I could not find my limbs," or "I lost all feeling in my limbs," or "I was afraid my legs would give way under me." The lower limbs do indeed sometimes give way in the first stages of the paroxysm of severe fright, extreme annoyance, or intense joy. Such muscular collapse may be considered as a reaction to an hallucination of the deep or muscle sense. In the instance of the accident to the school-teacher described, the fall may have been due either to such a reaction, to an hallucination of muscle sense, or to the second stage of the paroxysm, that of general rigidity. It may be remarked in passing, that the dropping of the upper limbs to the sides of the body which is the ordinary gesture of real or feigned surprise in common social intercourse, may have its origin in the normal epileptoid paroxysm—

53. Willbrand, H., and Saenger, A.: Neurologie des Auges, **3**:1006, part 2, 1906.

a gesture whose familiar meaning is meant to be readily recognized by the observer.

Vestibular hallucination—subjective vertigo—is a very frequent symptom in the first stage of the epileptic attack. It is sometimes the only manifestation of the disease for certain periods of time, as may be seen from Case 7 (p. 731). The sensation of vertigo or dizziness is perhaps the most frequent symptom of that reduction or disintegration of cerebral function, which precedes the muscular phenomena of the normal epileptoid paroxysm consequent upon exposure to sudden painful or emotional stimuli. Such popular expressions as "dizzy with joy," "dizzy with fright," or "dizzy with surprise," and a hundred other variations of the same expressions testify to the frequency of vestibular hallucinations in the first stage of the paroxysm associated with these normal conditions.

Epigastric hallucinations are exceedingly common. In the experiments to be mentioned later, the facial expression was frequently one of disgust, which sometimes persisted for a number of seconds. The reason for it given by the subject was that the procedure had made him or her "a little sick at the stomach." At other times the facial expression was one of uneasiness and the reason offered for this was a "peculiar sensation" of an indescribable nature within the epigastrium.

In cases of epilepsy in which this stage is of sufficient duration, automatism is frequent. An ordinary example of it may be seen in the following observation of Case 12 (p. 733) from the Vanderbilt Clinic Series:

> When requested to take off his shoes for the purpose of testing the Babinski reflex, he stared at the writer in an astonished manner for a moment, smiled foolishly and began to rub the back of his head in a persistent manner. He then turned away his face, smiling all the time, and making chewing movements. A minute later he rose from his seat and proceeded to walk. When half way across the room he turned around. His face was now expressionless. When led by the examiner to his seat, he offered no resistance. He then became rigid for a moment and a barely perceptible tremor ran over him. Gradually, in the course of two or three minutes, consciousness began to return and in another minute or so he was sufficiently recovered to respond to the request to remove his shoes.

Cases of epilepsy exhibiting far more complicated movements have been described by a number of investigators. One or two such cases from the writer's series will be given later.

In the case of the normal paroxysmal reaction to sudden emotional or painful stimuli, automatism may be observed with surprising frequency during the first stage, if the latter is sufficiently prolonged. The purposeless movements of the surprised or frightened person, which consist in rubbing the chin, in scratching the neck, in picking up objects and replacing them, in chewing movements, in walking

movements, in irrelevant smiling or giggling, and in the hundred other such unmotivated yet perfectly coordinated movements, are familiar to everybody and need no further description.

The analgesia which occurs during this and the subsequent stages of the normal epileptoid reaction will be dealt with later. Suffice it to say at this point that such insensibility to pain has the implication of a contraction of the field of consciousness.

2. *The Stage of Tonic Rigidity.*—This is the most constant as well as the most striking manifestation. It may be observed in cases of exposure to the sudden action of almost any stimulus whether sensory or emotional, from the mildest to the most intense.

The experiments carried out in this connection were of the simplest kind. The subject stood with his back to the observer. He was asked to relax his muscles as much as he could. The back of the concha of the subject's ear was then sharply struck by the observer's finger nail. In an instant he assumed a state of general tonic rigidity which was more pronounced in women and children than in adult men, and much more so under circumstances to be detailed later. The back was straightened, the head raised; the thighs and legs were extended, the shoulders were elevated, the upper limbs were extended and pronated; the fingers were either extended on the contracted palm or half closed in a partial fist; the thumb was adducted against the palm.

It will be remembered that a characteristic feature of the tonic rigidity of the epileptic fit is the adduction of the thumb against the palm in such a manner that the fingers are flexed over it. Gowers [54] calls attention to this fact. Spratling [10] mentions it as among the signs which differentiate the true epileptic fit from malingering. The position of the thumb in the condition of decerebrate rigidity described by Turner [55] was across the palm. In the previously mentioned case of tonic rigidity described by Stewart and Holmes, the flexed fingers covered the thumb. In one instance of the experiments in question the flexion of the fingers was complete, and the thumb of the hand observed was covered by the flexed fingers.

The sudden prick of a pin produced the same result, but the method is obviously disagreeable both to the subject and to the observer. The application of cold to the body, though remarkably adequate in evoking the epileptoid reaction in a number of persons, has been repeatedly tried and was finally discarded as difficult to apply with the needed degree of suddenness. The fact that a little time elapses before the subject experiences the low temperature of the tube (about half a

54. Gowers, Sir William R.: Epilepsy and Other Chronic Convulsive Diseases, 1901, p. 88.

55. Turner, C. Violet: A Case of Prolonged Hyperpyrexia in a Child with a Midbrain Tumour, British J. Child. Dis. **13**:261, 1916.

second), as well as the persistence of sensation after its removal, introduces confusing factors. No experiments were made with loud sounds or sudden flashes of bright light for the reason that the effects of these stimuli may be studied almost anywhere. Everybody who travels in public conveyances has noticed that the sound of a bursting tire impels a number of the passengers, particularly of the female sex, to leap for a moment into the characteristic attitude of decerebrate rigidity. Everybody has seen women drop objects which they hold in their hands on exposure to any one of the numerous stimuli comprised under the category of "surprise." Whoever will take the trouble to observe his own reactions at times when the highest functions of the nervous system are on the ebb, will find himself bounding into the decerebrate attitude on the slamming of a door, a clap of thunder, a flash of lightning, or a friendly slap on the shoulder from behind.

It goes without saying that the duration of these attacks is exceedingly brief—momentary. Yet brief as the attack may be, the sudden change of posture from that of relaxation to one of general rigidity is always strikingly pronounced.

The condition known as myotonia congenita is characterized by the tardy relaxation of normally contracting muscles. To persons who are subject to this affliction, the normal brief paroxysms of general tonic rigidity becomes a source of embarrassment and danger. The onset of the tonic attack is caused in such persons under the same conditions and with the same rapidity as in persons with a normal musculature, but the myotonic muscles, once contracted, are not relaxed with the same degree of despatch. The author has observed one such attack in a myotonic person, brought on by the application of a weak faradic current.[56] The rigidity which ensued lasted for two or three minutes and was singularly pronounced, the consistency of the muscles being more that of wood than of flesh. Thomsen[57] has described such patients as falling on exposure to relatively trivial emotional stimuli, and the cases described by Bell[58] are certainly of the same kind.

The most powerful of the emotional stimuli capable of inducing a state of rigidity in normal persons is by all odds that of fear. Such popular expressions as "scared stiff," "petrified with fear," and others of the kind, testify to the potency of fear in inducing a state of high and equal tonus in the antagonistic sets of muscles throughout the body. The state of sudden anger ranks in this respect perhaps only next to fear.

---

56. Rosett, Joshua: A Study of Thomsen's Disease, Brain **45**:1, 1922.

57. Thomsen, J.: Tonische Krämpfe in willkürlich beweglichen Muskeln, Arch. f. Psychiat. **6**:702, 1876.

58. Bell, Sir Charles: Quoted by A. McL. Hamilton in the Med. Rec. **29**: 85, 1886.

The hunting of deer at night by means of a jack-light is prohibited by law in a number of our states. The writer was informed by a colleague, who witnessed a deer exposed to the glare of a strong light at night, that the animal thus surprised stood stock still and could have been easily shot or taken in that condition. The writer himself has repeatedly seen rabbits by the roadside at night, in the glare of an automobile light, remain immobilized by the sudden action of the stimulus. Nor can it be said that animals who live in the sunlight are not trained to react in the presence of light. That their lack of training is with respect to the suddenness rather than to the nature of the stimulus applied, is borne out by the experiments on persons mentioned above. When the subject had been previously informed of what was to be done to him, no reaction, or only a very slight reaction took place.

The vast utility of the reaction of general rigidity on exposure to the sudden action of a stimulus will appear in a subsequent section.

The Paroxysmal Outcry: In the more pronounced instances of general rigidity induced in connection with the present study, the instant of onset was marked by an expiratory sound, "ah" or "oh"—the familiar sound of surprise. A similar outcry marks the onset of the tonic stage of the classical epileptic fit. In both cases it is caused by the sudden contraction of the muscles of the diaphragm, the abdomen and the thorax, and of those muscles which obliterate the opening of the larynx. The result is that a column of air is forced through the approximated vocal chords. The prolonged, unearthly cry of sudden intense fear is brought about by a similar mechanism. The ordinary "Ah!" or "Oh!" of pretended surprise in polite conversation almost certainly has its origin in the paroxysm of general rigidity ensuing in the momentary normal states of decerebration.

Anesthesia Associated with the Paroxysm: The inadequacy of stimuli to make themselves manifest as sensations in that state of reduced consciousness which is associated with the epileptoid reaction to "surprise" is very striking. Every physician in active practice can relate instances of patients who had received painful injuries during states of excitement and who were quite unconscious of any pain until some time later. The case of Livingston, who experienced no pain while his arm was bitten by a lion, is a classical example. Rivers [59] tells of the complete cessation of pain in the inflamed skin over his shins during excitement when the vessel in which he sailed was threatened with shipwreck. The writer himself had the following experience:

While suffering from an intense toothache in an isolated house in the mountains one early morning, he heard the cry of the cook downstairs that the

59. Rivers, W. H. R.: Instinct and the Unconscious, Cambridge University Press, 1922, p. 58.

oil stove was aflame. He ran down, put out the flame, then returned and proceeded to dress for breakfast. The idea of dressing for breakfast seeming strange, he recollected that some time earlier in the morning he had been annoyed by the thought that he would not be able to eat breakfast on account of the toothache. It then dawned on him that the intense pain had entirely disappeared. It was not before the expiration of fifteen minutes or more that the gradually returning pain attained its former intensity.

Similar experiences may be found on all sides and need not be dwelt on.

3. *Clonic Movements.*—In the experiments on the effect of the sudden application of a stimulus, mentioned above, the momentary state of general tonic rigidity was followed in about half the cases by clonic movements. They were observed in the hands and fingers and were of the choreo-athetotic type—a few movements of small amplitude and little force. In this respect, therefore, the experiments, in which the stimulus employed was of necessity very weak, are not conclusive. Far more definite was the occurrence of clonic movements following the state of rigidity which the writer has observed in the normal paroxysmal reactions in the ordinary experiences of every day life. A mere suggestion of the familiar circumstances under which such clonic movements take place is sufficient.

The behavior of the person who by accident drops a fragile object from his hands is quite characteristic. Following the momentary tonic spasm of the entire body musculature, the person executes a few movements of partial flexion and extension of the upper limbs at the wrists, elbows and shoulders. The momentary general rigidity which immediately ensues when a person has received even a slight burn is always followed by vigorous movements, which frequently involve the entire body and all the extremities. The trembling, and the movements of larger oscillation, following the rigid state in the case of exposure to the powerful emotional stimuli which bring about the states of fear and anger are always prominent. Darwin [60] describes the state of fear as follows:

"The word 'fear' seems to be derived from what is sudden and dangerous (H. Wedgwood; Dictionary of English Etymology); and that of terror from the trembling of the vocal organs and body. . . . Fear is often preceded by astonishment, and is so far akin to it, that both lead to the senses of sight and hearing being instantly aroused. In both cases the eyes and mouth are widely opened, and the eyebrows raised. The frightened man at first stands like a statue, motionless and breathless. . . . The mouth becomes dry, and is often opened and shut. One of the best marked symptoms is the

60. Darwin, Charles: The Expression of the Emotions in Man and Animals, 1873, p. 289.

trembling of all the muscles of the body. . . . As fear increases into an agony of terror, we behold, as under all violent emotions, diversified results. The heart beats wildly. . . . There is a death-like pallor; the breathing is labored; the wings of the nostrils are widely dilated; there is a gasping and convulsive motion of the lips, a tremor on the hollow cheek, a gulping and catching of the throat; the uncovered and protruding eyeballs are fixed or they may roll restlessly from side to side. The pupils are said to be widely dilated. All the muscles of the body may become rigid, or may be thrown into convulsive movements. The hands are alternately clenched and opened, often with a twitching movement . . . ."

The sudden desire to urinate, of the frightened or intensely surprised person, is proverbial. Urinary incontinence in women is not at all uncommon under such circumstances. The writer once had a housedog who urinated immediately and involuntarily each time he was threatened with punishment.

In the face of the familiar facts just mentioned, it will not perhaps be altogether far-fetched to call attention to the similarity of the muscular state of an attentive and "appreciative" audience and that of the muscles in the epileptic paroxysm. The muscular tension of an attentive audience is manifested by the rigid immobility of the individuals, as may be judged by the tense facial expressions and the fixed postures. It is a fact well known to political economists and historians that all social customs have their roots in physiologic and biologic phenomena. The custom of applause of an appreciative audience may not improbably have its origin in the physiologic redistribution of muscle tonus following its massive and equal distribution, the latter, in turn, being consequent on that suspension of a large portion of the cerebral functions which is implied in the act of concentration.

4. *The Stage of Recovery.*—The process of integration of the disintegrated cerebral functions in the last stage of the epileptic fit is marked by hallucinations, and the muscular reactions to these, similar to those of the first stage. Owing to the concomitant exhaustion, however, this stage is of longer duration than the first. Even when the first stage of a major attack of epilepsy is so brief that all subjective and objective manifestations of the progress of extinction of the cerebral functions are apparently absent, or when the aura is only momentary in duration, the last stage, that of recovery, is always of considerable length. The symptoms of the gradual restoration of the extinguished cerebral functions may in some cases be entirely masked by the ensuing exhaustion, and the process of integration take place during the profound sleep which follows, so that on awakening the patient may be merely dazed and stupid. In other cases, however,

when the exhaustion is not great, the gradual recovery of cerebral functions gives rise to those periods of massive supply of tonus alternating with periods of its redistribution, which are manifested in the so-called hysteroid convulsions. In still lighter attacks, automatism of a complicated kind makes its appearance. In one of the patients from the Vanderbilt Clinic series the first three stages of the attack were frequently insignificant and entirely masked by the fourth:

Case 3.—Mary L., aged 34, a Lithuanian was housemaid in an American family. She spoke good English and the chief complaint was of times when she appeared not to understand a word of English; and, in spite of the protests of her mistress, kept up an incessant jabber in her strange tongue. Of this, however, the patient herself was quite unconscious. One such attack occurred in a crowded street while she was walking with her brother. She carried a heavy hand-bag. When the brother noticed that she was behaving in a queer manner, he attempted to relieve her. She refused to part with the bag and continued to carry it for a number of minutes until she had recovered. She was unconscious of the entire procedure. On examination it was found that the attacks of automatism were always preceded by slight motor symptoms.

In the experiments spoken of in the foregoing sections, the first stage of reduction in the conscious state was not observed; the second, that of general rigidity, though very distinct, was exceedingly brief; the third, that of clonic contraction, was insignificant; but the fourth, though not as prominent as the second, was of sufficient duration to make its observation possible in a number of instances. It lasted for about a minute, during which the subject was plainly dazed and stupid and frequently executed a number of irrelevant coordinated movements.

In case of the more powerful sudden emotional or painful stimuli, the stage of exhaustion and recovery is always very pronounced. Everybody has seen women drop into the first chair, apparently exhausted, after the spasm of general rigidity ensuing on fright, or on the reception of a very bad or even of a very good piece of news. After exposure to the stimuli which induce the severer states of fright or anger, the exhaustion may be of many hours' duration, and automatism of a complicated kind may mark the recovery of the disintegrated cerebral functions. In such cases the attack may subsequently be repeated and thus enter the domain of true epilepsy, as in the following case, which came under the writer's observation in the Neurological Clinic of the Johns Hopkins Hospital, in the summer of 1914:

Case 4.—A healthy man, 33 years of age, complained of short lapses of consciousness on several occasions, extending over a period of some years. The last attack which was unusually long, impelled him to seek medical advice. Its history was: About a week before his appearance at the clinic, he went to a country store to procure supplies for his farm. He placed the purchases in his car. The next event of which he was conscious was sitting up in bed, a cup of coffee in his hand, and his wife beside him. The history of the interval,

of which he was unconscious, was obtained from the wife. Having loaded the car with supplies, he had wandered away into the woods where he had been discovered after a prolonged search. He was brought home and put to bed. He asked for food and drink and when it was given him, ate and drank in an apparently normal way. All the time, however, he appeared to be quite unconscious of his surroundings. The writer does not remember the total duration of the patient's unconscious state.

The history of the first attack is as follows: The patient was busy in the farmyard when someone called him to come at once into the house. He found the family crowding in a state of great alarm into the kitchen. There he discovered his father lying in a pool of blood, his throat cut and in convulsions. The young man said not a word and returned to the farmyard. A few minutes later he was discovered bothering about an old wagon. He had been quite unconscious from the moment that he saw his father dying.

The reason for the repetition of the paroxysm in such cases will be discussed later.

Instances in which the normal reaction to sudden painful or emotional stimuli is of such intensity that it cannot be distinguished from the classical major epileptic fit may be found throughout the extensive literature on epilepsy. A single example from Dr. Borie's [61] cases, collected in the innocence of the early part of the last century, will suffice as an illustration:

An English army officer fought a duel with a Parisian student. The latter was the first to fire. The bullet passed through the air at some distance from the Englishman's right ear. The officer immediately fell down in a "frightful attack of epilepsy."

THE BIOLOGIC AND PHYSIOLOGIC SIGNIFICANCE OF THE NORMAL EPILEPTOID REACTION

The manner in which, among a crowd of sensory impressions, a single one succeeds in dominating the sphere of consciousness as a sensation, frequently to the utter exclusion of all other impressions, is not entirely understood.

What is true of simple sensory impressions is likewise true of the emotions and of the processes of thought. A mother who sees her child in danger of being injured is for the time being relieved of any grief with which she may be afflicted. A person intent on the solution of a difficult scientific problem may be "thoughtless" regarding his social duties.

Moreover, sensory impressions, emotions, and processes of thought are in this respect interchangeable. Instances exemplifying the manner in which an emotion, by becoming dominant, shuts out the sensations of acute pain, have been mentioned in the previous section. The

61. Borie: Des maladies nerveuses en général, de l'épilepsie en particulier, 1830, p. 324.

reverse is likewise true. The sensation of a severe toothache may lift entirely the burden of an emotion. And a toothache or a sudden emotion may make it impossible for the scientist or the business man to continue a train of thought.

That a relation similar to that which exists between the processes involved in the sensations, emotions and thoughts, obtains also between all of these cerebral functions on the one hand and the purely automatic functions of the midbrain and the spinal cord on the other, we have already seen. Any one who is not specially trained, and who will attempt the experiment of inducing in himself a state of general muscular rigidity by conscious effort, will discover its inadequacy as compared to the action of even such a trivial stimulus as the sudden prick of a pin. The following experiment illustrates the incompatibility of the higher cerebral functions with the tonic function of the midbrain and the spinal cord:

An old friend of the writer's, Professor C. of Washington, D. C., a frail man near seventy, was fond of exhibiting a feat of muscular prowess which none of his younger and by far the stronger friends could achieve. With the back of his head and the heels of his feet supported on the edges of two chairs he would lie straight and stiff, with as many men sitting on him as his length permitted, for a number of seconds. His eyes during this performance were tightly closed and the facial musculature tense. In order to test his conscious processes during this state of rigidity, the writer once attempted to engage him in conversation on a subject to which he was particularly susceptible. He immediately shouted "Get off!" and dropped to the floor. Upon inquiry he admitted freely that any appeal to his intellectual processes was quite incompatible with the muscular performance.

From a biologic standpoint the importance of the dominance of certain nerve functions at the expense of others must be obvious. Of the large number of vague impressions which a person experiences while tramping idly through the woods, it is obviously of the utmost importance that the sensations or emotions experienced in the presence of a snake should become dominant to the exclusion of the sensations and emotions experienced in the presence of fruit or flowers. In the long course of natural selection those animals whose nervous systems were defective in this respect must have perished, both because they could not detect and obtain their food and because they could not escape from their enemies, with the necessary degree of efficiency and dispatch.

We have seen under the heading of "Anatomic and Physiologic Considerations," that since animals are constructed on the plan of a number of jointed segments, the measured movement of a distal segment is conditioned by the fixation of the joints of the relatively central segments of the body or the limbs. When an animal is exposed to the sudden action of a stimulus, it is of the utmost importance that every

relatively distal segment be in readiness to execute movement, as soon as the kind of movement and the segments that are to participate in it, are determined by an appraisal of the nature of the stimulus. Such determination of the nature of the movement by an appraisal of the stimulus is a function of time, and is therefore a variable; while the necessary preliminary to the possible needed *movement of any kind,* is a constant, and is not conditioned by the factor of time. The act preparatory to *any* needed movement of *any* relatively distal segment consists in the fixation of *every* relatively central joint. The entire organism is thus converted into a solid base from which a measured pull can be exerted in any direction. And this is the state of the general tonic rigidity into which the organism bounds the instant it is exposed to the action of a stimulus whose nature is, for the time being, undetermined. That this state of rigidity is simultaneous with a suspension of certain cerebral activities we have already seen.

So overshadowing are, on the whole, the benefits derived by the organism from this reciprocity between the conscious state and the function of tonus, that it has become established as a rather fixed mechanism. The latter may be inferred from the fact that in a function whose goal is obviously a temporary partial suspension of all neuromuscular activity, the initial stage, which is that of suspension of the higher cerebral activities, is nevertheless accompanied by a certain amount of muscular rigidity and frequently by clonic contractions. The function in question is that of sleep and will be considered later.

The normal paroxysm described in the foregoing pages may be observed throughout the vertebrate and the invertebrate world. It may be seen in man as well as in the deer and rabbit; it may be observed in the brief movement of retraction which precedes the forward movements of escape of the surprised frog as well as in the similar movement of the frightened housefly.

It is important to bear in mind in this connection, that in the course of organic evolution a number of the primary functions of the organism come to be utilized in secondary ways, in accordance with the new needs imposed by an ever changing environment. Along these secondary ways a function may attain such a degree of development as to overshadow its primary uses. Thus, the expiratory sound which results from the contraction of the thoracic musculature in the normal paroxysms of general rigidity, comes to serve in the social animal, endowed with organs for the perception of sound, the important use of a danger signal. The cry of the frightened man, the squeak of the bird, and the croak of the surprised bullfrog all subserve this secondary purpose. Another such secondary use of the normal paroxysm of general rigidity is exemplified by the posture of rigid immobility assumed by certain insects when exposed to a danger which may be escaped by feigning death.

THE EPILEPTOID REACTION ASSOCIATED WITH CERTAIN FUNCTIONS

In the performance of every function necessitating a powerful contraction of the abdominal muscles, the entire musculature of the body and the limbs bounds into a state of increased and equally distributed tonus. This equal distribution of tonus to antagonistic muscles immobilizes the joints of every body segment and converts the entire osseomuscular mechanism into a solid base from which the abdominal muscles can exert a powerful pull. The functions of defecation, of parturition, of the expulsion of mucus or of foreign bodies from the bronchi by coughing, the acts of vomiting and sneezing all depend for their performance to a greater or less extent on the contraction of the abdominal muscles.

We have seen that an increase of muscle tonus is conditioned by the suppression, complete or partial, of certain cerebral functions. This conditioning is exemplified in a striking manner in the acts of defecation, of sneezing, and of parturition.

The circumstances under which the act of defecation is best performed by civilized adults on the one hand, and by stupid persons, children and animals on the other, point to the extent to which its performance is dependent on the relinquishment of the highest cerebral functions. It is a familiar fact that civilized adults, in the performance of this function, seek isolation. The absence of external stimuli facilitates the reduction of the conscious state down to the level where the proprioceptive impulses can exert an unhindered action on the tonic mechanism. On the other hand, young children stand in no such need of isolation in the performance of this function. In them, the cerebral inhibitory mechanism is still unripe; while stupidity is but a manifestation of poorly developed cerebral functions. Such young persons and stupid persons may be considered as possessed of the nervous system of an animal in which the lower levels are adequate to regulate the state of tonus. This is exemplified by Weed's [37] decerebrate young kittens, a large percentage of which failed to exhibit the phenomenon of muscular rigidity.

It is a well known fact that the epileptic fit may be averted by the action of such stimuli as may enter the sphere of consciousness in the form of sensations. Some of these stimuli have been mentioned in the introduction to this study. The same stimuli have a similar action in interrupting to a greater or less extent the progress of that reduction in the conscious state which is essential to the tonic action of the muscles in the acts of defecation, sneezing and, sometimes, parturition. The ease with which the act of sneezing is prevented by any expansion of the conscious state, as for example by a remark from a bystander on the ludicrousness of the approaching act, is familiar to all; and every

obstetrician knows how the tonic action of the muscles of the parturient woman may be interrupted by the appearance of a visitor.

The symptoms of disintegration of the highest cerebral functions during the period of transition between the waking and the sleeping state manifest themselves by the hallucinations which we call dreams. The state of awakening, which is the reverse process—that of integration—is marked by the same hallucinations. Baillarger's [62] essay may be profitably consulted on the subject. Herbert Spencer [63] attempted to trace the origin of religious beliefs to the hallucinations occurring during sleep. The art of psychanalysis is utilizing these hallucinations in the discovery of the factors which determine the onset of some of the neuroses.

On the whole, authorities appear to agree respecting the activity of the muscular system during sleep. Duval's [64] statement is that with the disappearance of voluntary movement, reflex movements persist and become even easier of elicitation. Bertin [65] is certain that reflex movements are exaggerated in sleep. This investigator puts forth the remarkable idea that reflex movements would indeed occur with greater frequency in the sleeping than in the waking state if it were not for the fact that the condition of repose deprives the organism of that stimulation on which reflex movements depend. Dejerine [66] thought that the sleeping person is like the decerebrate animal; that spontaneous movement disappears, but that reflex movements persist and are perhaps elicited with greater ease. Graham Brown,[20] indeed, likens the state of decerebration to that of light sleep. Waller,[67] however, calls attention to the fact that in profound sleep the muscles are completely relaxed and the tendon reflexes suppressed. Rosenbach,[68] who studied the subject of sleep on young children, gives the result of his observations about as follows: In the beginning of sleep the child has a frowning expression of irritability, and the reflex susceptibility is increased. Somewhat later the reflexes are diminished, except the abdominal and the cremasteric reflexes, which persist. When fully asleep, the corneal reflex is absent, the pupils become contracted, the abdominal and the tendon reflexes are abolished.

The slight discrepancies in the conclusions of different observers are undoubtedly due to the fact that they were drawn from observations of different stages of sleep. The writer's own observations agree with

62. Baillarger, M. J.: Des hallucinations, etc., Paris, 1846, p. 476.
63. Spencer, Herbert: Principles of Sociology, Vol. 1, 1910.
64. Duval, M.: Sommeil, Dictionnaire Jaccoud, 1882.
65. Bertin, E.: Sommeil, Dictionnaire Dechambre, 1881, p. 266.
66. Dejerine, J.: Traité de pathologie général de Bouchard, **5**:377.
67. Waller, A. D.: An Introduction to Human Physiology, 1896, p. 571.
68. Rosenbach, O.: Das Verhalten der Reflexe bei Schlafenden, Zeitschr. f. klin. Med. **1**:358, 1880.

those of Rosenbach, with the exception of a phenomenon which occurs in a still earlier stage of sleep, which is not described by that investigator. The phenomenon in question may be observed with ease on a travelling companion who, in a sitting and uncomfortable position, begins to nod. The strain of the position as well as the process of nodding maintain such a person for a considerable length of time in a very early stage of the process of falling asleep. The facial expression of such a person is tense, drawn, or frowning, and agrees with Rosenbach's description of the child's expression in the early stage of sleep. The muscles of the limbs are undoubtedly spastic. That the spasticity is not due to the strained position may be ascertained in a child by permitting it to fall asleep in the observer's arms, or as a bedfellow. On one's dog the same observations may be carried out with ease. In any such observation on persons it is important not to arouse the subject's suspicion that he is being felt with any special end in view. for the result of such a suspicion is a state of awareness, which is certain to spoil the experiment. Dogs in the first stage of sleep, may be handled by their masters with a remarkable degree of freedom without being especially disturbed, and in such animals the observations may be carried out with a high degree of certainty.

Following this initial spasticity, the duration of which depends on the rapidity of the progress of sleep, a longer period succeeds during which clonic contractions are exceedingly common. These have occurred in every person of whom the writer has made inquiries, and in every such person they have been at times of sufficient strength to awaken him or her from sleep. This phenomenon is an example of the adequacy of the stimulus of movement in aborting the progress of the normal reaction. Such clonic contractions constitute in certain instances a serious obstacle to the function of sleep and cases of this kind have been described by Gowers.[69]

IV. THE PERIODICITY OF THE NORMAL EPILEPTOID REACTION

The periodicity of the epileptoid phenomena described in the last section is self-evident, such phenomena being associated with the performance of functions of a more or less regularly periodic nature.

The periodicity of those epileptoid paroxysms which have been described as reactions to external conditions, however, is not self-evident. The external conditions which evoke this reaction in the organism are purely accidental. Fright, anger, the prick of a pin, the blow of a hammer on the nail, the slamming of a door, a sudden flash of light, a burn received, and a thousand other conditions of daily life, which necessitate sudden movement or sudden attention on the part of the organism, all or nearly all belong to the category of circumstances whose occurrence it is impossible to foretell.

---

69. Gowers, Sir William R.: The Borderland of Epilepsy, p. 106.

Notwithstanding, however, the impossibility of foretelling any particular one of those conditions, their total number is so great that the occurrence of some one of them, though not of any particular one, may be foretold with certainty in the course of a given reasonable length of time. From a mathematical standpoint, therefore, the external conditions in question may, in their totality, be considered as a *constant* factor. The action of this constant factor on the organism is not always adequate to evoke an epileptoid reaction of sufficient intensity to make its presence readily observable. The reason for this variable effect of the action of the constant external factor is the changing state of the organism; and the particular state of the organism which is the most favorable for the occurrence of a pronounced epileptoid reaction is subject to more or less regular periods of recurrence.

It is a fact too well known to need elaboration in this place, that the several functions of the neuromuscular system, like all the other functions of the body, are subject to more or less regular periods of exaltation and depression; periods, for instance, when the threshold value to external impressions is lowered, alternating with periods of higher threshold values; periods of exaltation of the functions of memory, reason and judgment, alternating with periods of mental dulness; and so on. Some of these periods, either of exaltation or of depression, of certain nervous functions go hand in hand with certain other periodic functions of the body, of which the menstrual period in women may be taken as an example. Others again are the common resultants of the periodic alterations in the activity of a number of functions of the body, such as those of digestion, excretion, sleep, the sexual function, and others.

The entire subject will perhaps become less complicated when viewed from the standpoint of the elementary causes which are productive of the phenomena of periodicity. A consideration of these elementary causes, besides, will facilitate an understanding of the action of such sedatives and hypnotics as the bromids and phenobarbital in diminishing the frequency and severity of epileptoid and epileptic paroxysms, and of the reverse action of caffein. A brief consideration of these causes will not, therefore, be amiss.

In a perfectly stationary medium it might be expected that a body consisting of a single point, when acted on by two forces emanating from two other points, would proceed to move in a perfectly straight line along the diagonal of the parallelogram of forces. No stationary medium, however, exists, nor does a body without dimensions. Owing to these latter circumstances the line of movement of bodies is never a straight one. The simple reason is that at every point in the progress of the moving body its relations to the forces which keep it in motion are disturbed by the great number of other forces in the medium. The line of motion is, therefore, one of continual readjustment. It is an

undulating or a wavy line, such that the outline of each larger wave is marked by smaller undulations, and the outline of each of the smaller undulations is indented by still smaller ones, and so on as far as they can be perceived.

In the final analysis a function of the organism is the common resultant of a great number of movements. It is to be expected, therefore, that the course of each function should be that of an undulating line, made up of phases of exaltation and depression; that each such phase should be indented by smaller phases, and these in their turn, by still smaller ones. A concrete example is the ordinary experience that at certain times of the day mental vigor is at its best. While the latter is true from day to day, it is also true that, on the whole, the mental powers are more acute during certain seasons of the year. And while this holds good from year to year, every adult has experienced the long ebb and flow of his mental faculties extending over a period of some years. And yet even this last undulation is only one which marks the outline of that large undulation of the mental faculties, the separate phases of which extend over the periods of infancy, childhood, adolescence, young adult age, middle age, senescence, and senility.

There is one more theoretical point before returning to the practical subject in hand. It must be borne in mind that the degree of undulation —the height of each phase—other circumstances being equal, depends on the *rate* of motion. Any increase in the rate, implying as it does a more difficult adjustment of the line of motion, must result in larger periodic disturbances—in swings of larger amplitude. A slower rate of movement, on the other hand, permits the resumption of the disturbed balance between the forces which keep the body in motion at shorter intervals of distance, with the result that the undulating line approaches nearer the course of a straight line.[70]

In the application of this latter consideration to the concrete example given above, it will be seen that persons whose intellectual activities are normally sluggish, experience but little change in their mental function during different times of the day or during different seasons of the year. On the other hand, persons whose mental activities are intense experience these periodic changes in a marked degree.

If the course of the several functions of the nervous system is marked by periods of exaltation and depression, we can see why the constant external determining factors would be potent to bring about a pronounced epileptoid reaction only at certain times.

In the experimental production of this reaction described above, it was found to be much more pronounced in women during the menstrual period; on days when the weather was depressing than when

70. A familiar illustration is the vibration of the parts of the cheaper makes of the automobile after the machine attains a certain rate of speed.

it was bracing; and, generally, when the person was in a depressed physical or mental state than when vigorous and cheerful; and it was brought out with especial prominence under the action of a drug which intensifies the activity of the lower levels of the nervous system—under the action of caffein, to be presently described.

V. EXPERIMENTS ON THE EFFECT OF CAFFEIN, BROMIDS AND PHENOBARBITAL ON THE EPILEPTOID REACTION AND THE EPILEPTIC PAROXYSM

The conclusion of Rivers [71] and of Hollingworth [72] regarding the action of caffein is that under the influence of moderate doses, reactions are executed with greater despatch and correctness. Rivers, however, remarks that "when taken in excess, the stimulating action may be so transitory and followed by so great a decrease, that it may legitimately be spoken of as an accelerator of fatigue."

Hollingworth notes that, "small amounts of caffein tend to produce retardation in discrimination time, this retardation being accompanied by a great number of false reactions. The false reactions appear to be caused by a preliminary briskness produced by the caffein, and the retardation in the reaction time caused by a voluntary caution and the attempt to eliminate the false reactions. This is a test in which stimulation does not make for efficiency except after long practise."

The writer's experiments on the effect of this drug on the epileptoid reaction and the epileptic paroxysm, while corroborating the conclusions of the investigators just mentioned, have brought out some additional points. These experiments were undertaken with a view to determining whether the normal epileptoid reaction is essentially of the same nature, of similar origin, and subject to similar changes under similar conditions, as the epileptic paroxysm. In the approach to the solution of this problem an attempt was made to find an answer to the following questions:

1. What is the most obvious effect of caffein and of the sedatives on a vertebrate organism whose nervous system may be considered to correspond to the lower levels of the human nervous system?

2. What is their effect on the functions of the different levels of the normal human nervous system?

3. What is their effect on the subnormal nervous system of the epileptic?

In the choice of vertebrates of low organization the advantages offered by those having a smooth, hairless, linear body without appendages are obvious. Such animals are snakes.

---

71. Rivers, W. H. R.: The Influence of Alcohol and Other Drugs on Fatigue, Croonian Lectures, 1906, p. 50.

72. Hollingworth, H. L.: The Influence of Caffein on Mental and Motor Efficiency, Arch. Psychol. **3**: No. 22, 1912.

EXPERIMENTS ON REPTILES

The experiments [73] were conducted on black snakes and on garter snakes, altogether fifteen in number. The results were quite uniform and the description of a single experiment will apply to all the others.

Six snakes were fastened by strips of adhesive plaster to a table parallel to each other and at some distance apart. One piece of the strip was passed around the neck, another midway between the two ends and the third about five inches from the aboral end. Two snakes were injected with sodium bromid, ¼ grain (0.015 gm.) and ½ grain (0.03 gm.) respectively to the pound of body weight; two with the same amounts of caffein citrate, and two were left for control. Ten minutes later the six reptiles all lay quietly without exhibiting any tendency to struggle or to move in any way. The table was then gently tapped once with the finger. The bromidized and the normal snakes remained perfectly quiet, while the two caffeinized snakes exhibited a remarkable reaction. It was a wave-like movement whose outline was marked by a vibration which passed with great rapidity from head to tail. The duration of the entire reaction was about half a second; it then subsided completely. A stronger tap on the table produced the same reaction in a more pronounced form—the amplitude of the movement was greater. The reaction was more pronounced in the reptile which had received the larger dose. By repeated tapping of the table one played on the two reptiles as on a stringed instrument, except that the vibrations in this case were not audible but visible.

When touched gently near the tail end, the control and the bromidized snakes made no response, while the caffeinized snakes responded by a brisk movement. A stronger touch brought out some movement in the normal snakes. The same movement in the bromidized snakes, though of smaller amplitude, was of somewhat longer duration; and the movement was most pronounced and briefest in the caffeinized reptiles.

The experiments on these animals with other drugs and after mutilation, are not relevant to the subject in hand and will not, therefore, be discussed.

The obvious answer to the first question, then, is that caffein enhances to a very marked degree the capacity of the lower levels of the central nervous system for the reception of the simpler and more common kinds of stimuli and the response to these by simple reactions. The action of the bromids is the reverse of that of caffein.

SUBJECTIVE EXPERIMENTS

The question respecting the effect of these drugs on the functions of the several levels of the central nervous system seemed difficult to answer from purely objective experiments. The writer, therefore, undertook to experiment on himself, with the following results:

The action of caffein in more than moderate doses (caffein citrate 15 grains [1.0 gm.] three times a day, in addition to three cups of

---

73. The writer wishes to express in this place his gratitude to the administration of the New York Zoological Gardens, and especially to Mr. Ditmars, the curator of reptiles, and to Mr. Moony, the keeper, for their liberality and courtesy. The abundance of material and the amount of assistance which these gentlemen placed at the disposal of the writer left nothing to be desired.

coffee) was to enhance to a very marked degree the susceptibility to all simple impressions, and the response to such impressions by simple automatic reactions. The epileptoid reaction manifested itself in the author in a striking manner on exposure to kinds of stimuli which had never evoked this reaction before. The sound of shutting a door, the unexpected sight of an acquaintance, the momentary idea that an object resting on the edge of a table might drop to the floor, and any of the numerous conditions which cause the sudden arrest of attention, or which demand sudden movement, brought on a momentary state of general rigidity, and a subsequent period of exhaustion and "bewilderment." The typewriter was operated with greater facility and despatch, the exercise of handwriting was made more rapid, and so were all acquired automatic movements. While reading was much faster, the contents did not ingrain themselves with the necessary clearness. The coherence of a train of thought was very markedly diminished, its elements succeeding each other with great rapidity. After four days the matter read appeared devoid of meaning; coherent thinking became very difficult and a state of excessive alertness and hyposomnia set in, which necessitated the discontinuance of the drug.

The action of bromids (30 grains [2 gm.], three times a day) was in certain respects the reverse of that of caffein. The capacity for the perception of simple impressions was markedly blunted, and all simple reactions became sluggish. During the six days when the drug was taken manifestations of an epileptoid reaction were not once perceived. Reading became difficult and the matter read left a hazy, indistinct impression. Thinking, although sluggish and difficult, was not, however, marked by the incoherence which had made the process defective when caffein was taken.

The action of phenobarbital was, on the whole, the same as that of the bromids. It appeared to the author, however, in his subjective experiments, that phenobarbital (3 grains [0.2 gm.] four times a day for four days) had, in addition, a rather disagreeable effect on the circulatory system, as judged from slight cyanosis of the finger tips and shortness of breath on exertion. The latter action, however, may have been due to a special susceptibility.

After taking caffein, the highest integrative nerve functions failed for two reasons. One was on account of the unusual crowding of external impressions. The other was because attention was continually distracted by involuntary reaction to each impression in the crowd. The distinguishing feature of the highest functions of the nervous system is a response by a single reaction to an accumulated number of separate impressions, after the latter have been integrated in a definite manner. The immediate response to each separate stimulus received was, therefore, in itself incompatible with these highest activities.

The reason for the action of the bromids and of phenobarbital in diminishing the capacity of the highest nerve functions, on the other hand, appeared to be the tardiness with which impressions followed each other, the paucity of those which entered the domain of consciousness as definite sensations and emotions.

THE EFFECT OF CAFFEIN AND PHENOBARBITAL ON THE EPILEPTIC MANIFESTATIONS

As the most pronounced effect of caffein was to produce a wide-awake state of the kind described, it appeared that the epileptoid reaction manifested must be essentially different from the epileptic paroxysm. The muscular phenomena of the first appeared to be brought about merely by an increased alertness; those of the second are undoubtedly preceded by a suspension or reduction of the conscious state. The experimental administration of caffein to epileptics was, therefore, approached with a sense of the possibility of substituting a pronounced normal epileptoid reaction for the epileptic paroxysms. The doses of caffein given were, however, small, the largest being 3 grains (0.2 gm.) four times a day, the smallest 1 grain (0.06 gm.) at bedtime. The cases included every possible category of the epileptic disease: organic as well as nonorganic, inherited and noninherited, major, minor and psychic. The following ten cases, picked out at random, give a fair idea of the effect of the three drugs on the disease:

CASE 5.—J. P., a boy, aged 17, of robust, healthy appearance, presented no apparent stigmata. Birth, infancy and childhood had been normal and he had been normal at school. No familial history of epilepsy was elicited. Five months before his appearance at the clinic, he struck his head against an iron banister and was unconscious for twenty minutes. The scalp was not bruised. A few hours later there occurred a major epileptic fit, the first he had ever had. Two days later there was a second major fit and after that fits occurred at irregular intervals, on the average five a week. He had been taking drugs for some weeks, which diminished the number and severity of the fits. Minor fits only had occurred for the last two or three weeks on the average of one a day. During these the face was drawn to the left side.

Roentgenograms of the skull were negative. The reflexes were normal and so were apparently the internal organs. The mentality was dull and tardy; memory was very poor. Relatives stated that before the onset of the attacks the boy had been very bright.

The patient was given caffein citrate, 3 grains (0.02 gm.), four times a day. Five days later he reported eleven major attacks and a great number of minor ones. Mental dulness was very much increased and confusion of ideas was so great that he had great difficulty in understanding what was said to him. Caffein was discontinued and phenobarbital, 1 grain (0.06 gm.), three times a day, was given instead. A week later great improvement was reported. No attacks had occurred and mental dulness had to a great extent disappeared. In the next three weeks the patient, still taking phenobarbital, reported one minor attack.

CASE 6.—A. P., a girl, aged 6, had the appearance of a normal child, except for abnormally long ear lobules, inherited from her mother. There had been a difficult instrumental delivery, but development was normal. Minor fits had been observed for the last eighteen months on an average of once a day. No hereditary history of epilepsy was elicited. The tendon reflexes were slightly increased on the left side; there was no Babinski sign, but the right toe went down with greater constancy than did the left on plantar stimulation. The abdominal reflexes were active and equal.

The child was given phenobarbital, ¼ grain (0.015 gm.) once a day, which reduced the number of fits to one in two days. Two weeks later, caffein citrate, ½ grain (0.03 gm.) was given three times a day. Eight days later it was reported that she had had one or two fits every day since last seen. The child was again given phenobarbital, ¼ grain (0.015 gm.) twice a day, with the result that the fits occurred once in two days.

CASE 7.—Wm. A., a lad, aged 18, gave a history of normal birth; he had been delicate as a child, but development was normal. At the age of 6 he fell from a swing and struck his head badly, but there were no immediate serious consequences. The record of his school career was very poor. In the last two years he has had altogether two or three major attacks. Minor attacks have occurred on the average twice a week. The minor attacks consist at times merely in a "sensation" or feeling of dizziness. A niece of the patient has epilepsy and is feeble minded.

Examination of the labyrinths made by Dr. Rosenbluth, disclosed no abnormality. The reflexes and internal organs were normal. The mentality was dull and sluggish.

The patient was given phenobarbital. Two weeks later he reported one attack of dizziness and one "sensation"; no loss of consciousness. Phenobarbital was discontinued and caffein citrate given instead, 1 grain (0.06 gm.) every four hours. Eight days later he was reported as feeling generally brighter in mind. Only one attack of dizziness had occurred. Within the next two weeks there were a very great number of attacks of dizziness (exact number not known). Caffein was discontinued and phenobarbital given instead. A week later he was reported as having had no attacks. The mental condition was as dull as before caffein had been taken.

CASE 8.—S. R., a woman, aged 27, had a definite facial asymmetry, the left side being fuller and sagging. The left palpebral fissure was larger than the right. There was no history of epilepsy or insanity in the family.

Major epileptic attacks had occurred since infancy. At the age of 14 these became much more pronounced and occurred about once a week. She had not menstruated until the age of 24, when she came to the Vanderbilt Clinic. There, she was placed first on bromids, then on phenobarbital. She menstruated soon after. Attacks after that recurred once a month, during the menstrual period.

The reflexes were normal. She could not voluntarily turn the mouth to the left, nor could she close the left eye without at the same time closing the right.

She was given caffein citrate, 3 grains (0.2 gm.) four times a day. Three major fits took place within the next four days. Caffein was discontinued and phenobarbital was given again. Within the next month she had one attack, during the menstrual period, as before.

CASE 9.—L. S., a woman, aged 44, of normal appearance, gave no direct hereditary history of epilepsy. Nocturnal attacks of major epilepsy appeared five years before, without known cause, and had recurred on the average of one every eight months. Attacks of motor aphasia of about one hour's duration had also been observed on the average once a month, for the last five

years. For the last two or three years the right upper limb had become paralyzed for a few minutes nearly every day. Of late the left arm, too, had become affected in the same way. She complained of failure of memory and of difficulty in thinking.

She had been taking bromids and phenobarbital for the preceding three years; there had been no major attack within the last twelve months.

The blood Wassermann reaction and roentgenograms were negative. The right tendon reflexes were somewhat exaggerated.

The patient was given caffein citrate, 3 grains (0.2 gm.) four times a day. A week later she reported as feeling generally brighter and more cheerful. She had not the "dopy" feeling that she had before caffein was given, but had had difficulty in falling asleep. Caffein, 3 grains (0.2 gm.) was then given once a day only. Three days later she had eight major attacks in one night. She was unconscious all night and most of the following day. Caffein was discontinued and phenobarbital therapy again instituted. A month later she reported herself as feeling the same as before caffein had been taken. There had been no major attacks.

Case 10.—F. S., a man, aged 30, except for a slight facial asymmetry, appeared normal. He had a hypospadias. There was no history of epilepsy in the family.

Eight years before, the patient had been for a time under unusual stress, working all day and attending on his invalid mother at night. One night, when especially fagged out, he had a nocturnal major fit, which had been repeated on the average once every six months until eighteen months before. For the last eighteen months he had been taking drugs regularly and had not had any fits. He complained of mental dulness and sluggishness and of a feeling of great fatigue on the least exertion. The reflexes and internal organs were normal.

Phenobarbital therapy was discontinued and caffein citrate, 3 grains (0.2 gm.) four times a day, was given instead. For the next ten weeks he reported weekly. A great improvement was noted in his general condition. The mental dulness and sense of fatigue disappeared and he became bright and cheerful. For the first time in his life he felt like a "real man." An occasional momentary feeling of dizziness was experienced, which caused no discomfort. A singular change took place in the patient's character. He was no longer the same modest young man as before caffein was given; he was boastful and vainglorious. The caffein was reduced to 3 grains (0.2 gm.) once a day, given early in the afternoon.

Case 11.—R. R., a woman, aged 18, appeared normal. At the age of 5 months a series of convulsions extending over a period of three days was observed. Otherwise infancy and childhood were normal. There was no history of epilepsy or of insanity in the family.

Minor attacks had occurred on the average of one a day, for the last three years. She had been taking phenobarbital for about twelve months, and during that time, had had three or four attacks during menstrual periods only.

The reflexes and internal organs were normal. The mental status was normal to ordinary tests. She had an insuperable dislike for any kind of mental effort and had no intellectual interests of any sort. She could not bear to read, or even to see a movie. On the contrary, she had a great passion for manual labor, and worked rapidly and indefatigably from morning to night and enjoyed it. She had a quick and violent temper.

The patient was given caffein citrate, 3 grains (0.2 gm.) four times a day. Within the next five days she had twenty-six minor attacks. Caffein was discontinued and phenobarbital given instead.

CASE 12.—H. L., a man, aged 22, appeared normal except for a large and very ugly nose. Of thirty-two known blood relatives, not one had epilepsy. Infancy and childhood were normal.

Major and minor attacks had been observed for the last six years. During the first three years the major attacks recurred about once a week but after that time he had taken drugs regularly and had not had any major attacks for a number of months. Minor attacks took place on the average of one in two days, during which there was marked automatism. The reflexes and internal organs were normal.

Phenobarbital therapy was given for seven weeks. In this period there were no major attacks and only four minor ones weekly. He was given caffein citrate, 3 grains (0.2 gm.) four times a day. A week later he reported that he had had six minor attacks. Within the following five days the patient had three major and a number of minor attacks and he appeared demented at times. Caffein was discontinued and phenobarbital given once more.

CASE 13.—R. W., a man, aged 22, appeared healthy and well built. He had a facial asymmetry and large, congested hands. Both traits were inherited from the mother's side of the family. An aunt on the mother's side and a grandmother both had epilepsy.

Birth, infancy and childhood were normal, but he had a rather poor school career. About three years before he began to have nocturnal major attacks, without definite determining cause. The attacks were repeated about once a month. The reflexes and internal organs were normal.

The patient was given phenobarbital and the attacks ceased. Caffein citrate was then given. A week later he reported two nocturnal attacks and complained of irritability and restlessness. Caffein was discontinued and phenobarbital therapy instituted. No attacks occurred during the next two months.

CASE 14.—G. W., a boy, aged 11, appeared normal, except for a notable difference in the size of the two ears, the left being the larger. The latter characteristic was inherited from the father. No epilepsy or insanity was discovered in the family. The father, however, suffered from "nervousness" to such an extent that he had had to abandon his occupation.

Birth, infancy and childhood were normal. Minor epileptic attacks had been observed for the last three years. The attack sometimes amounted only to a "sensation." At other times, however, the right arm was raised over the head and became very rigid; at the same time the patient's face turned to the right, and his body rotated in the same direction. At still other times, the attack consisted in automatic coordinated movements, such as running about the room, etc. The reflexes were normal and the internal organs healthy.

The patient had been taking phenobarbital for some months, with the result that the attacks had become very light and recurred about once a week. He was placed on caffein citrate, 1 grain (0.06 gm.) three times a day. Seven days later he was reported as having had four very severe attacks. Two of these were major—the first major attacks he had had. The drug was immediately discontinued and the patient was once more placed on phenobarbital.

The action of these drugs may now be summed up in the most general terms:

It will be remembered that the amplitude of oscillations in a line of motion depends on the rate of the motion. In terms of force, therefore, the action of caffein was to increase the amplitude of oscillations of the course of certain nerve functions by an acceleration of the rate

of their motion. The bromids and phenobarbital, on the other hand, by retarding the rate of the movement, diminished the amplitude of oscillation of those functions and made their course nearer that of a straight line. Exogenous and endogenous stimuli, therefore, were at an advantage in evoking reactions in the first instance and at a disadvantage in the second. A chronic state of sluggishness of nerve function induced by the action of the bromids or phenobarbital is the price paid by the epileptic person for his rescue from the danger of a paroxysm.

VI. THE RECIPROCITY OF NERVE FUNCTIONS. THE IDENTITY OF THE EPILEPTIC PAROXYSM WITH THE NORMAL EPILEPTOID REACTION

The reciprocal mechanism for the innervation of antagonistic muscles has been dealt with in a previous section. We have seen, too, that a similar reciprocal action is to be found at higher levels of the central nervous system. Prominent among the latter are the tonic centers of the spinal cord and midbrain on the one hand and of the cerebrospinal and cerebropontine systems on the other. At still higher levels, we found that a similar reciprocity exists in the domain of the sensations, the emotions and of thought.

If, now, we make a general division of the nerve structures into low—those which receive impressions and respond to them by simple immediate reactions—and high—those which inhibit and integrate—we shall likewise find them to be subject to the general rule of reciprocity of nerve functions—we shall find that the active functioning of one set of structures is simultaneous with an inhibition of function in the other. The incessant restlessness of the dog or the monkey with the ablated cortex indicates that the functioning of this highest nerve structure in the intact animal is concomitant with an inhibited activity of the lower nerve structures. And the same fact is in another way exemplified by the action of large doses of caffein on the normal human being. That action we have seen to be a great enhancement of the lower nerve structures for the reception of and the response to impressions; and such an effect we have seen to be incompatible with the exercise of the highest functions of integration and of discrimination.

Mention was made in a previous section of the fact that certain primitive and dormant mechanisms in the lower levels of the nervous system may awaken to action when the higher and more efficient mechanisms, subserving similar functions, are removed. The statement must now be amplified by calling attention to the fact that such primitive mechanisms have for their salient characteristic the ready reception of simple stimuli and the immediate response to them by muscular movements of a simple pattern. If a transient disintegration of the highest functions is accompanied by an enhancement of the lower kinds of reactions, what must we expect when the highest struc-

tures of the brain are in any way permanently defective? In that case we may not only expect the enhancement of the lower order of reactions which result from the lack of a higher control, but, in addition to that, a resurrection of a number of ancient and long dormant mechanisms in the lower levels of the nervous system. The enhancement of the lower order of reactions must then be out of all proportion to the defect of the highest structures.

The latter consideration enables us to understand the extraordinary effect of small doses of caffein on the epileptic paroxysm. The same intensification of the lower nerve functions by caffein which, in the normal person, results in an exaggeration of the normal epileptoid reaction must, in the case of the epileptic person, set going a series of mutually aggravating conditions. A vicious circle is created in which, with each degree of enhancement of the lower nerve functions, there is an increased disintegration of the higher; and each degree of increased disintegration of the higher functions, in its turn, brings about an enhancement of the lower order of functions. In such cases, therefore, we are prepared to find the most pronounced manifestations of the epileptoid reaction. In these patients the effect was manifested by an exaggeration of the epileptic paroxysms.

We have seen, however, that the difference between the epileptoid reaction and the epileptic paroxysm is merely one of degree of intensity. We have brought examples of cases in which the normal epileptoid reaction could in no way be distinguished, even with respect to intensity, from the epileptic paroxysm, except that in the latter case the paroxysm was repeated. The reason for such a stability of the severer form of the reaction abides in those permanently enhanced capacities of the lower levels of the nervous system, which are concomitant with some permanent defect of the highest structures. Whether the defect is inherited or acquired makes no difference. Hauptmann[74] who made a study of fifty-two patients in whom epilepsy seemed to make its appearance as a result of exposure to war conditions, was able to trace, in nearly every instance, some pre-existent defect of the highest nerve functions. In the case of the accident to the school teacher and in Case 4, we see examples of epileptoid and epileptic reactions which approach the borderline from either side. The fact that the father in Case 4 committed suicide in a particularly shocking way indicates the probability of a permanent inherited defect of nerve structure in the son, and accounts for the recurrence of the vastly exaggerated epileptoid reaction.

In about half the total number of epileptics, the paroxysms are nocturnal. In the process of falling asleep and of awakening from

74. Hauptmann, A.: Ueber Epilepsie im Lichte der Kriegserfahrungen, Zeitschr. f. d. ges. Neurol. u. Psychiat. **36**:181, 1917.

sleep, the nerve functions exhibit the effect of a generally depressing condition; the highest functions are to a great extent extinguished, while the lower are still active. Such a state furnishes every circumstance for the release of the mechanism involved in the epileptoid or the epileptic fit: the disintegration of the conscious state, with the consequent freedom of action of the tonic mechanisms of the midbrain and the spinal cord. In the state of awakening the highest structures are last to recover. Here, too, we have the conditions necessary for the production of the fit: the full capacity of the lower nerve functions, coincident with the still depressed high cerebral activities. We have seen that the normal process of falling asleep exhibits the epileptoid reaction. In case of a subnormal structure of the brain, therefore, the process of falling asleep and of awakening must be very favorable to the onset of an epileptic paroxysm. The strikingly frequent occurrence of the epileptic paroxysm in sleep is, therefore, in exact accord with the other facts pointing to the essential identity of the normal epileptoid reaction and the epileptic paroxysm.

The character of the pronounced epileptic is beyond a doubt abnormal. Even that enhanced docility of temperament, which is frequently hailed as a rare virtue, must, both from a neurologic and a sociologic standpoint, be considered a defect. Such extreme toleration of the vices and weaknesses of one's fellows as is displayed by Dostoyevky's [75] Prince Mishkin, makes, in the long run, for the downward progress of the individual as well as for that of the race.

Extreme docility, however, is not incompatible with outbursts of wild rage. Case 12 possessed unusual docility and sweetness of temperament. Yet this patient complained of fits of rage and intolerance which impelled him on frequent occasions to commit violence on his fellows.

After months of study of Case 10, the writer came to the conclusion that the character of that patient was normal. After the patient had taken caffein for two or three weeks, the writer was one day astonished to hear him break out in a stream of boastfulness and vainglory. He boasted of the size of his genitals and of his prodigious sexual powers. On investigation both claims proved to be untrue. The defect in the faculty of discrimination became manifest when the disintegration of the highest faculties, brought about by caffein, was added to that which was inherent.

After a similarly prolonged study of the character of another patient of the Vanderbilt Clinic Series, the conclusion arrived at was that he was a model young man. He worked steadily, was devoted to his parents, spent his evenings at home and never went in bad company. He was a devout and regular attendant at church; he had never had an affair with a woman, being sincerely convinced of the

75. Dostoyevsky, F.: The Idiot.

sinfulness of illicit relations. One day he admitted being an habitual liar, a thief and a traitor. He was in the habit of lying on the least provocation. He had stolen money from his parents and from his relatives. He had robbed a cash register, though he did not really need the money. He had stolen an implement in his place of business. The object was soon missed and, in the patient's presence, a friend of his was accused of the theft. The result of the whole affair was that the real hero of the occasion was considered, more than ever before, the model of an honest, modest and capable employee.

The other patients yielded more readily a knowledge of the profound defects in their characters. It must be said, however, that these defects, far from being defects of instinct, as Clark [12] puts it, were rather the result of a defective inhibition of the instinct. Every one of the faults of character of these patients was obviously due to a tendency to react immediately and mechanically to environmental stimuli. They lacked that faculty of integrating impressions received, which makes itself manifest in the exercise of judgment and discrimination.

Viewing character as the sum total of the reactions of the human organism, the character of the epileptic person is closely correlated to his disease. This disease consists, as we have seen, in an enhanced capacity for the manifestation of the normal epileptoid reaction, brought about by a corresponding incapacity of the highest cerebral functions. In terms of "character," such an incapacity implies a relative inability to exercise the faculty of judgment; while the enhanced capacity for the immediate response to environmental stimuli implies, in the same terms, a release of the lower animal instincts.

Viewing epilepsy from the standpoint of the mechanism and the cause, as outlined in the foregoing pages, a rational mode of prevention and of therapy will suggest itself to the thinking physician; and this must constitute the subject of a separate publication.

VII. SUMMARY

The investigation discloses a physiologic reaction in normal man and animals which consists of the following connected train of phenomena:

1. A temporary reduction, disintegration or extinction of the cerebral functions.

2. A tonic contraction of the entire skeletal musculature, with a certain predominance of the extensors, adductors and pronators; the consequent fixation of the joints resulting in the posture characteristic of the state of decerebrate rigidity.

3. Clonic muscular contractions, i. e., alternating contractions of antagonistic groups of muscles.

4. Recovery, marked by symptoms of integration of the disintegrated cerebral functions and a state of general exhaustion.

This reaction, in a more or less pronounced form, normally occurs under two sets of conditions: (1) on exposure to the action of a stimulus requiring sudden movement on the part of the organism, or the narrow focusing of attention, and (2) in connection with certain functions, such as sleep, defecation, sneezing, coughing, parturition.

The mechanism of this reaction is to be found in certain anatomic structures and physiologic functions underlying the production and distribution of muscle tonus in the nervous system.

The biologic purpose of the reaction is the automatic fixation of the relatively central joints preparatory to any possibly needed movement of relatively distal segments of the body and limbs.

In the history of any individual organism this reaction occurs with greater frequency and in more pronounced form at certain times than at others. In other words, the reaction is characterized by a periodicity of occurrence.

The phenomena which constitute the normal reaction described, being in a great number of particulars the same as those which constitute the epileptic paroxysm, a suitable name for it would appear to be *the normal epileptoid reaction.*

The normal epileptoid reaction is elicited with greater ease under the influence of the same agencies which cause an exaggeration of the epileptic paroxysm. An example of such an agency is the action of caffein. On the other hand, influences which are potent in holding the epileptic paroxysm in abeyance, such as the action of the bromids and phenobarbital, have the power to diminish the manifestations of the normal epileptoid reaction.

In numerous cases it is impossible to determine whether the paroxysm belongs to the category of the normal reaction or to that of the epileptic disease. Mild cases of epilepsy and instances of the normal reaction of the severer grades are in every respect alike. The normal reaction may, however, be of such severity as to be indistinguishable from the major epileptic paroxysm.

The cause of the generally greater severity of the epileptic paroxysm is to be found in a neurologic principle which underlies the production of the normal epileptoid reaction—that of the alternating reciprocity of function of the higher and lower levels of the nervous system. A slight and transient suspension of the higher functions, resulting as it does in a release of function of the lower levels, brings about the normal epileptoid reaction. Any factor, therefore, which operates permanently so as to cause large periodic suspensions of the highest nerve functions, whether that factor be a brain tumor, a poison in the blood, a peripheral irritation or an inherited defect, will, by a permanent exaggeration of the normal reaction, tend to degrade it to the clinical picture of the epileptic paroxysm.

LESIONS OF THE OPTIC CHIASM AND TRACTS WITH RELATION TO THE ADJACENT VASCULAR STRUCTURES *

TEMPLE FAY, M.D., AND FRANCIS C. GRANT, M.D.
PHILADELPHIA

It is of unusual interest to note how few gross deformities are found at necropsy to explain the variety of visual field defects caused by suprasellar and interpeduncular tumors. Pressure on the visual pathways is the generally accepted cause, but how and where it is exerted is often a matter of speculation. That only 13 per cent. of the 148 cases of tumor in this area collected by Uhthoff [1] showed gross lesions of the tracts, is significant. As pointed out by Bartels [2] and Cushing,[3] there is usually little at necropsy to explain the complete hemianopias so often associated with these lesions.

The great destruction of the optic tracts seen in a recent case, caused by a large interpeduncular tumor, and the relationship of the offending adjacent vascular structures are of decided interest, and an attempt has been made in this case to correlate the visual field defects with the gross anatomic picture.

It is our purpose to present a case which showed deep notching of the optic tracts by the carotid arteries caused by pressure against them from an interpeduncular tumor. A search has been made for similar cases, and where diagrams existed they have been reproduced in order to throw further light, if possible, on visual field defects produced by such lesions.

There are four structures in this area which may exert direct pressure against the visual pathways in the presence of a large invading suprasellar lesion: the dural band connecting the anterior clinoid processes, the internal carotid arteries, the anterior cerebral arteries and the anterior communicating artery. The anatomic relations of these structures may be of interest.

The carotid artery, after leaving the carotid canal, passes forward through the middle lacerated foramen with the sixth nerve and the cavernous sinus, to a point just below the anterior clinoid process. Here it bends upward and backward in horseshoe fashion, to reach a position just below and to the outer side of the chiasm proper. It

* From the clinic of Dr. Charles H. Frazier, University Hospital, Philadelphia.

* Presented before the Philadelphia Neurological Society, April 28, 1922.

1. Uhthoff: Graefe-Saemisch. Handb. d. ges. Augenheilk. **11**:1262.

2. Bartels: Ueber Plattenepithelgeschwülste der Hypophysengegend (des Infundibulums), Ztschr. f. Augenheilk. **16**:407, 1906.

3. Cushing, Harvey, and Walker, Clifford B.: Chiasmal Lesions with Especial Reference to Homonymous Hemianopsia with Hypophyseal Tumor, Arch. Ophth. **47**:119, 1918.

then passes upward almost at a right angle, about 1 cm. behind the chiasm, almost touching the optic tracts, and quickly divides above into its terminal branches (Figs. 1 and 2).

The anterior cerebral artery passes forward over the optic tracts and chiasm as it ascends and converges toward the midline to pass up between the frontal lobes.

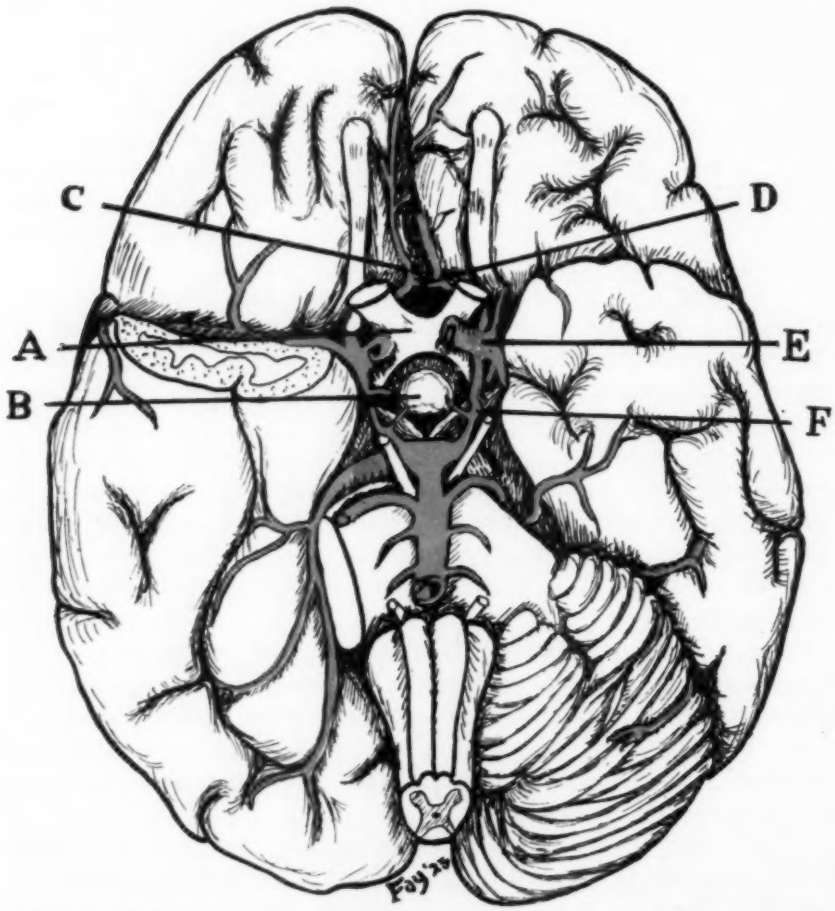


Fig. 1.—Relation of the arteries to the optic chiasm and pituitary on the base of the brain: *A*, optic chiasm; *B*, pituitary gland; *C*, anterior cerebral artery; *D*, anterior communicating artery; *E*, internal carotid artery; F, posterior communicating artery.

The anterior communicating artery is usually given off at a point just above and in front of the chiasmal decussation. The artery itself is small and seldom over 1 cm. in length. The posterior communicating arteries flank the interpeduncular space and lie well to the outer side of and below the optic tracts, and hence produce no influence on these structures.

The vascular structures that may be concerned in the presence of interpeduncular tumors are therefore the internal carotids, the anterior cerebrals and the anterior communicating arteries. Cushing [3] points out that there may be constriction of the nerve by a dural band connecting the anterior clinoid processes and cites a case of this character (Fig. 3).

HISTORY

It may be of interest to note that attention was first called to this type of lesion by Türck [4] in 1852. He reported a case of tumor of

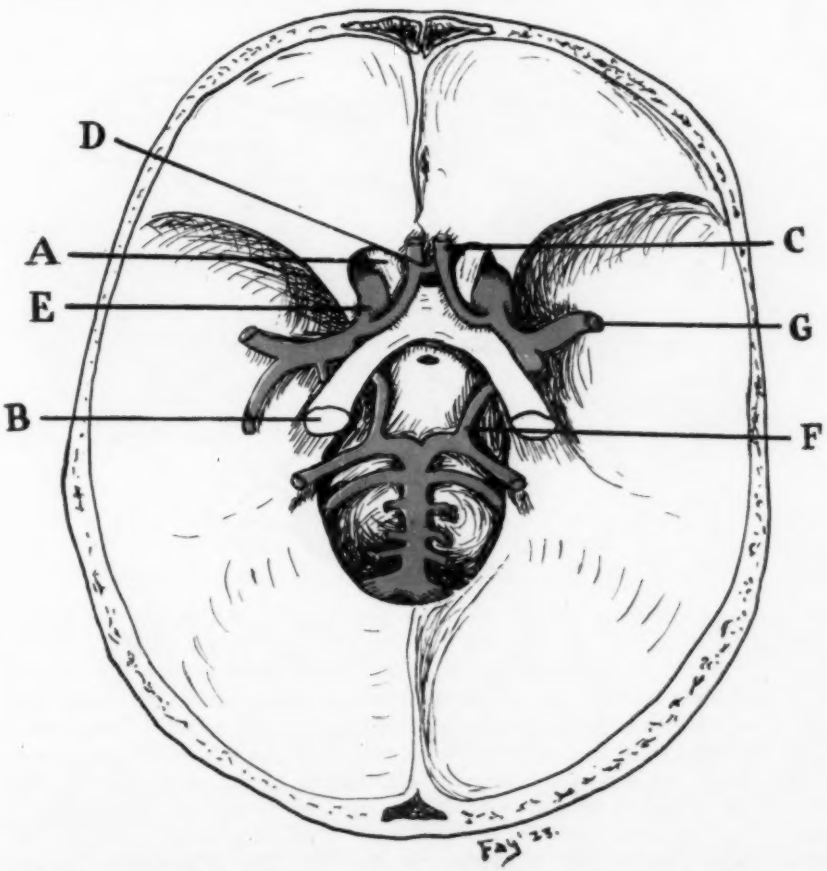


Fig. 2.—Relation of the arteries to the optic nerves, chiasm and tracts: *A*, left optic nerve; *B*, left optic tract; *C*, anterior cerebral artery; *D*, anterior communicating artery; *E*, internal carotid artery; *F*, posterior communicating artery; *G*, middle cerebral artery.

the hypophysis in which both nerves were deeply notched transversely by the anterior communicating artery; and he stated that the tract was also notched laterally on the left, but does not say by what vessel. The left nerve was so deeply cut that few fibers remained intact. The

4. Türck: Ztschr. d. k. k. Gesellsch. der Aertz. zu Wien **8**:299, 1852.

right nerve showed the inner third uninjured. There is no report concerning the fields in this case, but a note made one week before death stated that the left eye was completely amaurotic, while the right eye was only amblyopic. Sachs,[5] in reporting the findings in forty-eight necropsies which revealed hypophyseal tumors, twice found constriction of the optic nerves by the anterior cerebral arteries. In one of his cases, the tumor presented itself in front of the chiasm. Erdheim [6] has collected seven cases of optic tract and nerve constriction. Two cases showed the tracts almost cut in two, 1 cm. behind the chiasm,

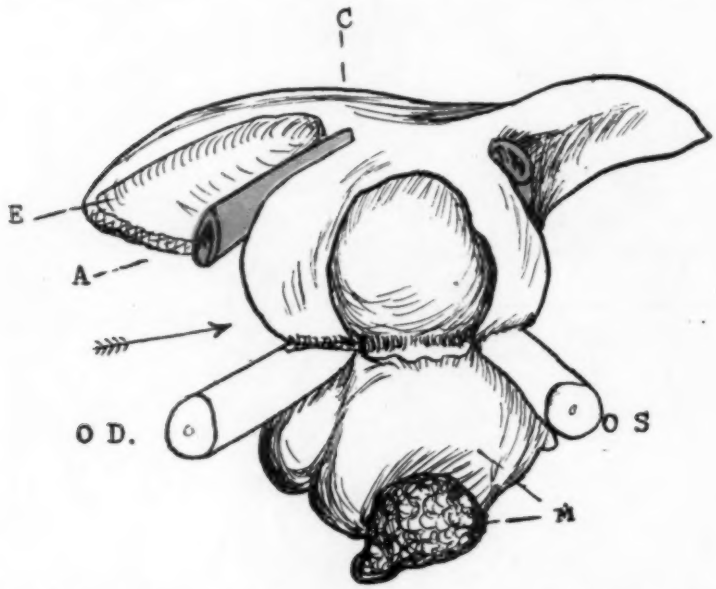


Fig. 3.—Front view (copied from the article by C. B. Walker and Harvey Cushing, Arch. Ophth. **47**:119, 1918) of the distortion of the optic chiasm and nerves produced by a hypophyseal adenoma which extended into the cranium. The right optic nerve is deeply cut by pressure against an artery. Both optic nerves have been compressed at the level of the arrow by a band of dura which connected the widely separated clinoid processes. *M*, the intrasellar tumor mass; *C*, the chiasm on top of the tumor; *O D* and *O S*, the right and left optic nerves; *A*, an artery deeply embedded in the right nerve at the chiasm; *E*, extension of the tumor into the temporal lobe. The visual fields presented "left homonymous hemianopia, incomplete in the left eye, with decided central scotoma in the right eye and contraction of the lower nasal quadrant."

from pressure by the carotids. The remaining five cases showed notching of the nerve or chiasm by the anterior cerebral and anterior communicating arteries. Visual fields do not accompany the reference. Uhthoff,[1] in reviewing the literature on hypophyseal disease for more

5. Sachs: Ztschr. f. Augenheilk. **13**:378, 1905.
6. Erdheim, cited by Bartels.

than 100 years, up to 1911, reports a case of vascular constriction of the optic nerves by the anterior cerebral arteries. The visual fields showed a bitemporal hemianopia, with contraction in the lower nasal fields. It is possible that the latter defect was caused by these arteries cutting the upper and outer tracts of the nerve which supply the lower nasal fields. Bartels,[2] in an extensive contribution to the pathology of this area, also reports a case in which pressure of a tumor from behind had pushed the chiasm forward, and in which the optic tracts were deeply notched by the anterior cerebral arteries. He points out that, owing to the location of the lesion, it is readily overlooked.

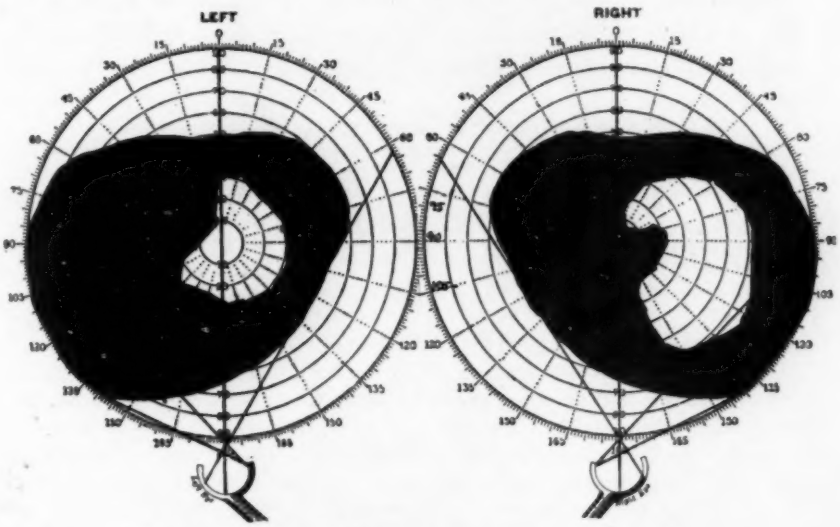


Fig. 4.—Visual fields from a case reported by Cushing.

De Schweinitz and Holloway,[7] in 1912, were the first to point out (in English) the rôle that these lesions might play in the production of some of the visual field defects seen in pituitary cases. These authors include in this paper a comprehensive review of the literature.

Cushing,[8] in his monograph on the pituitary and in six later papers [9] on visual field disturbances in cases of brain tumor, does not emphasize the defects caused by vascular lesions.

The visual fields in one of his cases showed an incomplete left lateral homonymous hemianopia, with marked lower nasal defect on the right side. There is a large cecocentral scotoma passing upward

7. De Schweinitz and Holloway: A Clinical Communication on Certain Visual-Field Defects in Hypophysis Disease, with Special Reference to Scotomas, J. A. M. A. **59**:1041 (Sept. 21) 1912.

8. Cushing, Harvey: The Pituitary Body and Its Disorders, Philadelphia, J. B. Lippincott Co.

9. Cushing, Harvey: Brain **44**:241, 1922. References to all papers are given.

from the outer lower nasal quadrant (Fig. 4). The notching by the artery shown in the drawing seems to impinge at a point where the very tracts that supply this area of the field should pass, and is possibly the cause of the defect in that portion of the field.

Hirsch [10] in a recent survey of the literature, reports the ocular findings in forty-five cases, and contrasts them with those of Uhthoff,[1] whose series contained 148 cases. He reports a large hypophyseal tumor that flattened out the chiasm and pressed it against the anterior cerebral artery, which was found to have deeply notched it transversely. A sketch from his drawing is given in Figure 5.

Walker and Cushing,[11] in 1918, reviewed 271 cases of their series, in 148 of which visual field deformities were present. It is of interest to compare the findings of Uhthoff, Cushing and Hirsch, as to visual disturbances.

VISUAL DISTURBANCES WITH PITUITARY TUMORS

Author	No. of Cases	Bitemporal Hemianopia	Homonymous Hemianopia
Uhthoff	148	37.0%	2.8%
Cushing	148	31.7%	14.8%
Hirsch	45	77.0%	0.7%

REPORT OF A CASE

History.—J. R., a white man, aged 31, was admitted Dec. 4, 1921, to the service of Dr. Frazier at the University Hospital, complaining of headache, vomiting and failing vision. He was well until July, 1921, when he began to have severe headaches, paroxysmal in type, both frontal and occipital. In October, his vision began to fail, and he found that he could not read small print. Soon after this he began to vomit, usually in the mornings, but this was not projectile in character. He noted occasional diplopia, but has had no convulsions or ataxia.

Examination.—Physical examination revealed a rather heavy and obese adult in a semistuporous condition, who complained of severe headache. There was no weakness of any cranial nerve. The eyegrounds showed 4 diopters of swelling in the disk on each side. The visual fields were slightly contracted, especially along the temporal margins of the upper portions (Fig. 6). The reflexes of the right upper extremity were increased, and there was marked increase of the reflexes in the lower limbs on both sides. Bilateral abortive ankle clonus was present, but there was no Babinski sign. The spinal fluid pressure was increased to 18 mm. of mercury. The blood and spinal fluid Wassermann tests were negative. The roentgen ray revealed a "top normal" sella (Pancoast). The tentative diagnosis was: increased intracranial pressure, probably from a tumor in the region of the sella. Visual hallucinations became marked.

Operation and Course.—Five days after admission, the patient's condition was such that it was necessary to perform subtemporal decompression. This resulted in improvement.

10. Hirsch, O.: Ztschr. f. Augenheilk. **45,** No. 5, 1921.

11. Cushing, Harvey, and Walker, Clifford B.: Studies of Optic Nerve Atrophy in Association with Chiasmal Lesions, Arch. Ophth. **45**:407 (Sept.) 1916.

December 19, and again January 9, a ventriculogram was made (F. C. G.), and a large defect in filling was shown to be present in the posterior horn of the left ventricle.

January 5, the visual fields (Fig. 7) showed that, in the month since admission, he had developed a left lateral homonymous hemianopia in spite of the decompression. Marked ataxia with increase of reflexes on the left side, numb-

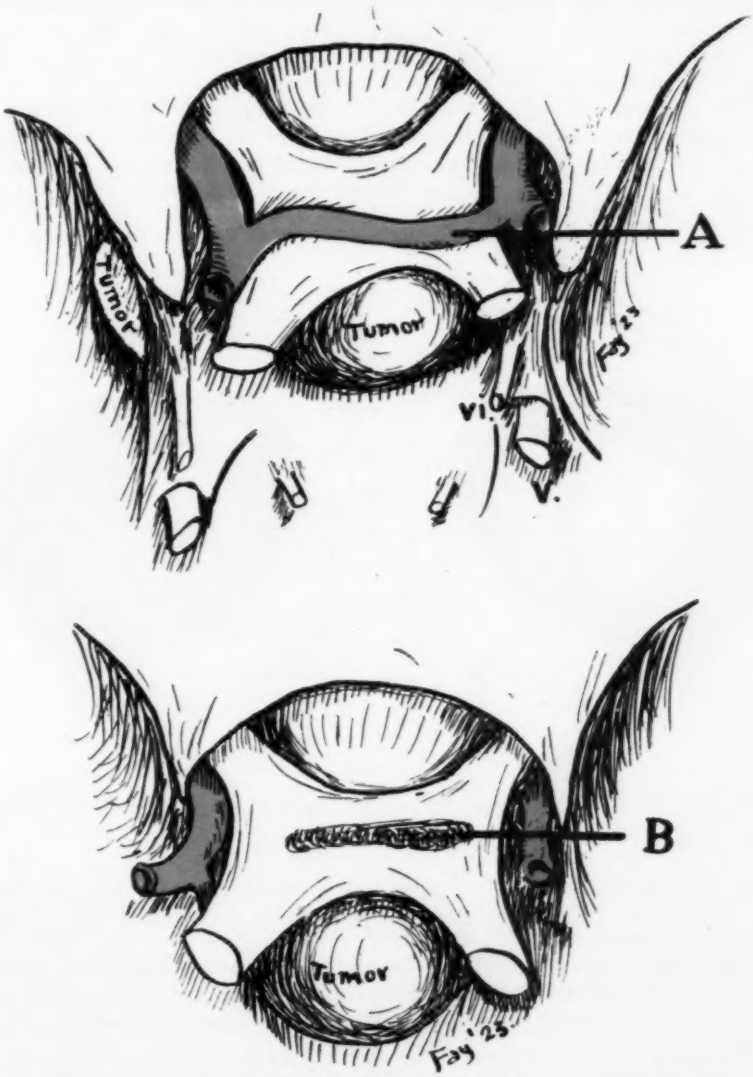


Fig. 5.—Notched chiasm viewed from behind (copied from the report by Hirsch): *A*, anterior communicating artery; *B*, notching of the chiasm by pressure against the anterior communicating artery.

ness, loss of the sense of position, dysmetria, and adiadokokinesis were also present.

Based on these findings, a parieto-occipital exploration on the right side, seemed indicated. The patient was operated on February 3, but no tumor was

disclosed. He made a good recovery, and was up and about the wards for almost a month, but he often dozed, and was aroused with difficulty. He gradually became more stuporous, and finally died on March 23.

Necropsy Examination.—Necropsy revealed a tumor, microscopically an adamantoma, about the size of a walnut, and apparently entirely suprasellar. It was lying in the interpeduncular space just posterior to the chiasm, which had been pushed well forward. The tumor extended into the third ventricle, and posteriorly as far back as the upper margin of the pons (Fig. 7). There was some apparent flattening of the optic tracts and chiasm by pressure from the tumor just below, but on closer inspection, after exposing the sylvian fossa by retraction of the lower borders of the temporal lobes, the optic tracts were found to be notched by the carotid arteries on both sides. On the left side, a deep groove was made on the outer and lower aspect of the tract by the internal carotid artery, 1.2 cm. behind the chiasm. Over the upper and outer portion of the tract, there was a shallow groove caused by pressure

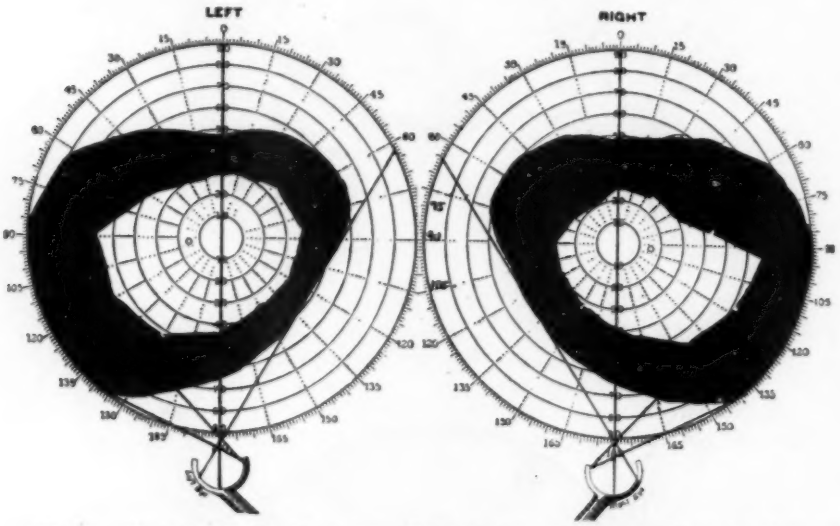


Fig. 6.—Visual fields (with 3 mm. disk) in the case reported, at the time of admission.

against the left anterior cerebral artery as it coursed forward over it. About the center of the chiasm itself, there was a slight dimpling of the structure where the anterior communicating artery exerted pressure. The right optic tract was greatly affected, the fibers having been cut almost two-thirds through by the right internal carotid artery. There was some herniation at the point of decompression, and the cerebral structures had been displaced slightly to the right.

COMMENT

From the visual field charts (Figs. 6 and 7) of this case we may make some interesting speculations.

A general shrinkage of the visual fields was apparent when they were first taken at the time of admission. There was a suggestive upper bitemporal slant which has been so often spoken of by Cushing and is so characteristic of hypophyseal lesions (Fig. 6). The day after these

fields were taken, subtemporal decompression was performed on the right side. Shortly afterward, the ventricles were emptied for a ventriculogram. Twenty-eight days after the first fields were taken the charts showed definite left lateral homonymous hemianopia, which had developed in spite of the decompression (Fig. 7).

Study of the specimen (Figs. 8 and 9) shows that the right optic tracts were so deeply cut by the carotid artery on that side, that only about one third of the tract remained at this point. It may have been that with the relief of pressure on the right, due to the subtemporal decompression, the tumor grew along the lines of least resistance, pressed the structures toward the right and the decompression opening. This would stretch the artery, and since its exit from the cavernous sinus allows but little lateral excursion, we may imagine that the softer

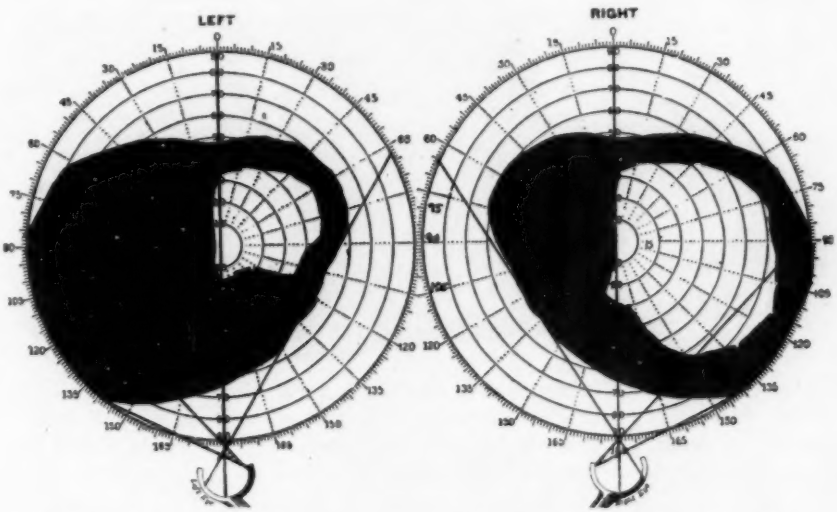


Fig. 7.—Visual fields in the case reported, twenty-eight days after admission and subsequent to ventricular tappings.

structures of the right optic tract lying between the tumor and the pulsating vessel were crushed against it by the enlarging growth, producing an almost complete functional block of that optic tract, and hence lateral homonymous hemianopia on the left side.

The left tract was not only cut by the carotid, but the groove formed was extended forward by the anterior cerebral artery which pressed against the structure as it coursed forward to form the circle. The tract had been caught in the elbow of this branching and the deformity was produced from pressure by the growth from below and medially. Whether the vessel was pulled across the optic tract as a result of the pushing of the structures to the right when tension was relieved by the decompression, with the consequent dragging of the left anterior cerebral artery over the tract on the left, cannot be

determined. Some mechanical factor caused notching of the tract and chiasm by the anterior cerebral artery on the left but not on the right (Fig. 9). It may well be, of course, that the tumor grew more on the left side, and direct pressure was greater here than on the right tract. In either case a left lateral homonymous hemianopia was produced within a month (Fig. 10).

It is of interest to note that the chiasm in this specimen does not ride high on the top of the tumor, but has been pushed forward; and there has been a great deal of pressure exerted on its posterior and lower aspect, with some notching by the tumor. There is no doubt that the fibers have been put on a stretch, and as pointed out by Cushing,[11] and reported by de Schweinitz [12] and Holloway, it is here that the papillomacular bundles suffer most.

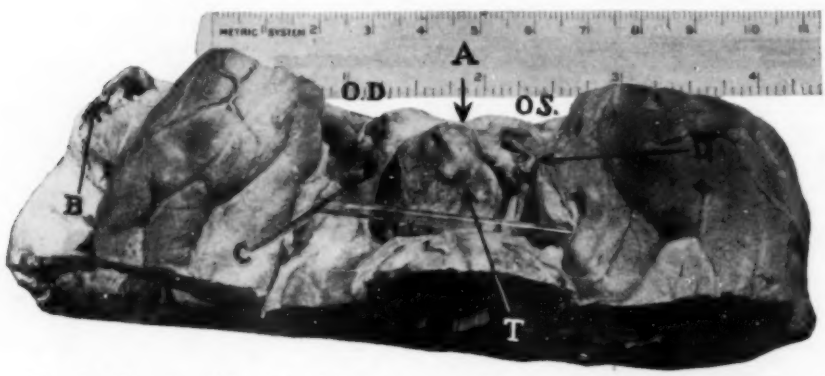


Fig. 8.—Tumor with optic chiasm, tracts and nerves from the case reported: *A*, the chiasm; *B*, ventricular hernia at the site of the subtemporal decompression; *C*, deep notching of the right optic tract; *D*, the carotid artery and the notched left optic tract; *T*, the tumor; *O D* and *O S*, the right and left optic nerves.

CONCLUSIONS

1. The situation of the optic nerves, chiasm and tracts makes them easily susceptible to injury from surrounding vascular structures in the presence of a rapidly growing tumor in the interpeduncular space.

2. With careful perimetry, defects of the lower nasal quadrants may indicate that lateral pressure is causing physiologic block of the visual pathways; the adjacent and overlying arteries may be responible for the field defect.

3. Lesions of this character, owing to their concealed position, may easily be overlooked at necropsy.

4. The offending vessels are the internal carotids, the anterior cerebrals and in several cases the anterior communicating artery.

12. De Schweinitz: Concerning the Evolution of Some of the Visual Phenomena of Pituitary Body Disorders, address delivered at opening of Peking Union Medical College, Peking, China, September, 1921, not published.

DISCUSSION

Dr. Charles H. Frazier: This case was confusing because the lesion was of pituitary origin, and yet the patient had a marked papilledema, which we do not usually associate with pituitary lesions. It was a case in which the ventriculogram proved misleading, since the distortion of the ventricle proved to be on the side opposite that of the lesion. There are many cases in which the only, or at least the most important, symptoms are disturbances of the visual fields, and I do not think that we have given sufficient consideration

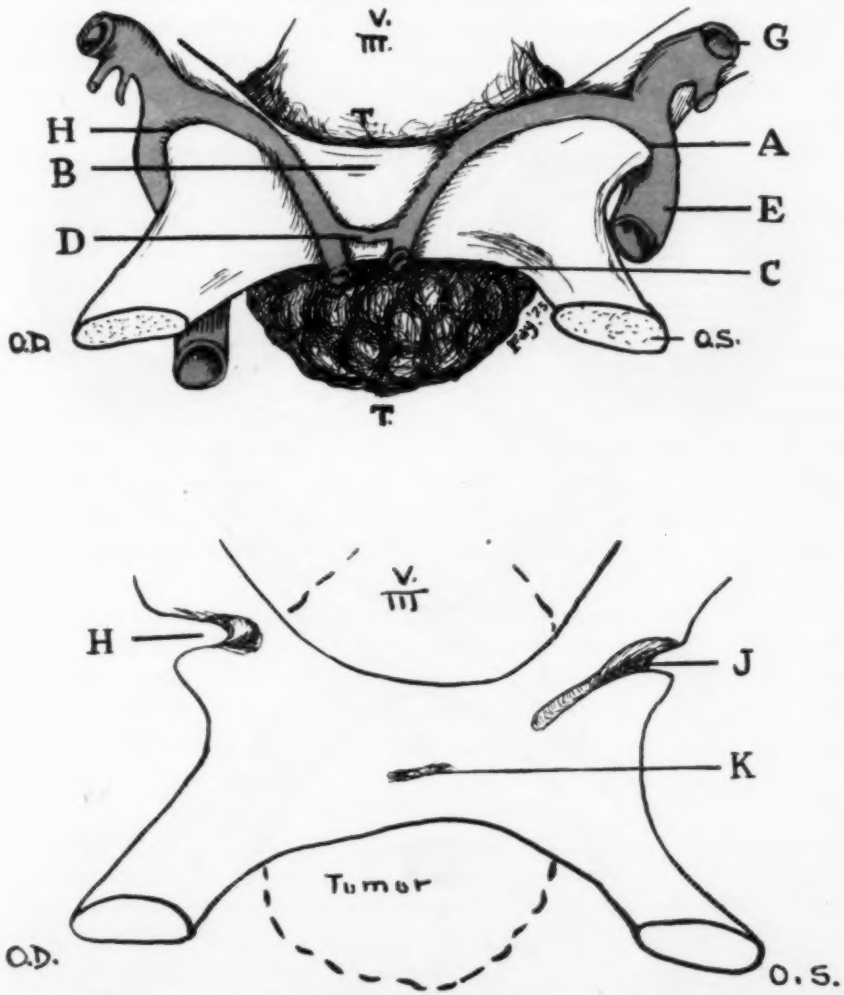


Fig. 9.—Chiasm and optic tracts of the case reported, viewed from in front, illustrating the distortion caused by adjacent arteries. *T*, tumor; *A A*, constriction of the optic tracts by the internal carotid arteries; *B*, the optic chiasm; *C*, anterior cerebral artery; *D*, anterior communicating artery; *E*, left internal carotid artery; *G*, middle cerebral artery; *O D*, and *O S*, right and left optic nerves; *H*, large notch caused by the right internal carotid; *J*, notch caused by the left internal carotid; *K*, depression caused by the anterior communicating artery.

to the part played by the surrounding vessels in the causation of field distortion. If I am correct, the observations which Dr. Fay has made in regard to the effect of the internal carotid artery are original, at least as to the contributions from this country.

Dr. Francis X. Dercum: This communication recalls a case described a number of years ago in which the clinical features were reported by S. Weir Mitchell, and the ophthalmologic findings by Dr. William Thomson, while I described the specimen. The patient presented during life the symptoms of a bitemporal hemianopia, and necropsy revealed an optic chiasm which had

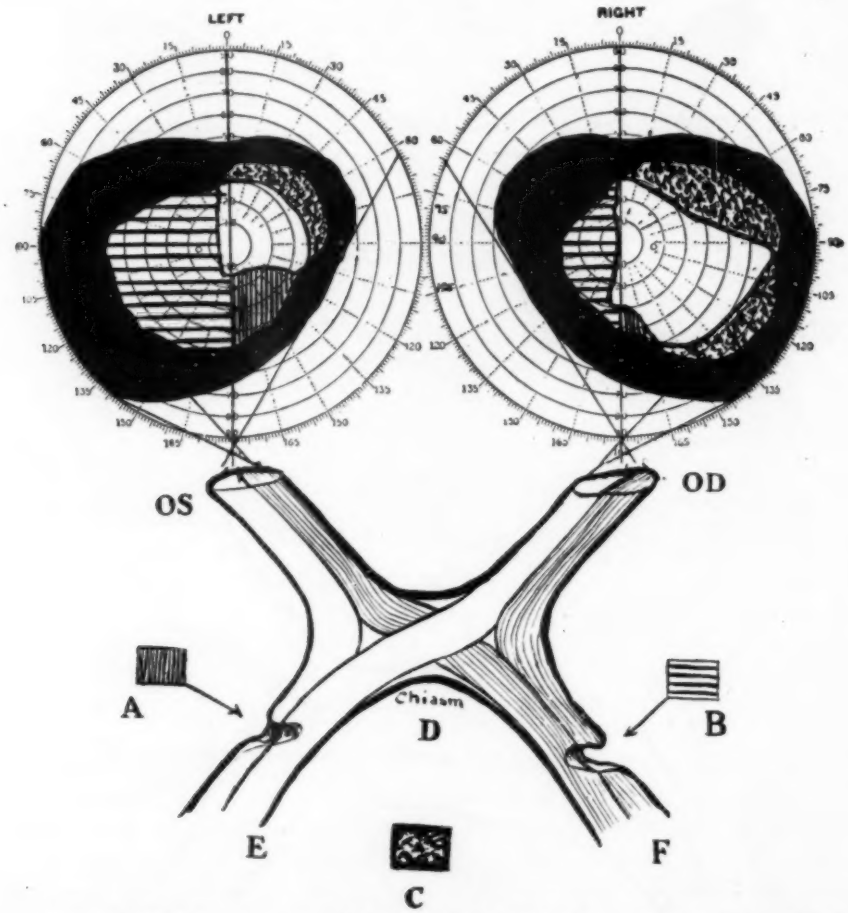


Fig. 10.—Effects of the notching of each optic tract, and of the decompression, on the fields of vision. Interpretation of shading: *A*, defect caused by left internal carotid and anterior cerebral arteries; *B*, defect caused by right internal carotid; *C*, field expansion following decompression; *D*, chiasm; *E* and *F*, left and right optic tracts; *O S* and *O D*, left and right optic nerves.

been divided in the median line by a slowly growing aneurysm of the anterior communicating artery. The divided strands of the chiasm and the optic tracts lay on either side of the aneurysm. The case was an excellent demonstration of the functions of the chiasm. The report was published in the *Journal of Nervous and Mental Diseases*, January, 1889.

EPIDEMIC ENCEPHALITIS

A PATHOLOGIC STUDY OF FIVE CASES INCLUDING TWO WITH MYOCLONIA

M. E. MORSE, M.D.

BOSTON

A systematic and detailed study has been made of five cases of epidemic encephalitis which have come to necropsy at the Boston Psychopathic Hospital. The aim of this work has been predominantly topographic rather than descriptive. In the earlier pathologic studies, attention was naturally focused on the midbrain; later researches have revealed that the changes in the nervous system are more widespread than was at first apparent. Recently these outlying lesions have received more study and the name "neuraxitis" has been proposed by French writers as more truly expressing the conditions found. Two of the cases in this series were of the myoclonic form, on which there are as yet few complete pathologic reports.

The cortical areas examined in each hemisphere were: the first frontal, the precentral, postcentral, temporal, calcarine, and hippocampal. Four or five blocks were made from the corpus striatum of each side, and two each from the thalamus and hypothalamus. Practically the whole of the midbrain, pons and medulla were cut into blocks. Three and frequently more levels of the cord were examined. The cranial nerves, the gasserian and posterior root ganglions, and the peripheral nerves were examined whenever they had been removed. The stains used were cresyl violet, Mallory's phosphotungstic acid hematoxylin and scharlach R. Several attempts were made with Alzheimer's methods for degeneration products and ameboid glia, but they were unsatisfactory as the tissue had been hardened in formaldehyd solution. The trunk organs and the pituitary were also studied in each instance.

REPORT OF CASES

CASE 1.—W. B. A., a man, aged 38, an electrician with a past history that had no bearing on the present illness, developed, March 23, 1922, a bad head cold. Four days later he suddenly became confused, spoke of violet rays coming through his body and feared that they would kill him. March 29, he became restless and had to be held in bed. He would put his hand to the back of his neck as if in pain. At no time was there any complaint or indication of double vision. March 30, he was admitted to the Psychopathic Hospital. His temperature then was 101.4; pulse, 116. He did not cooperate in the examination. There was no obvious defect in the cranial nerves; the pupils reacted well to light; gait and station indicated general weakness; there was slight cyanosis of the hands; the knee and ankle jerks were active and equal, as were also the triceps and abdominal reflexes. Examination of the chest and abdomen revealed no special abnormality.

Course.—During his stay in the hospital his behavior was in general of the katatonic type. He occasionally cooperated and answered questions, but for the most part he was uncooperative or negativistic; at times he was actually resistive. He frequently assumed rigid attitudes. He admitted that he experienced auditory and visual hallucinations, but he did not react to them. Underlying this behavior there was, however, a severe general intoxication. He became rapidly weaker with marked muscular relaxation, slight cyanosis and an increasingly weak and rapid heart. April 3, there appeared to be a divergent strabismus. He died April 5, the duration of the illness being twelve days.

Case 2.—E. P., a young French woman, aged 26, a teacher in a private school, with a past history that is not of interest in the present connection, was vaccinated Nov. 23, 1920, and had a good reaction five days later. November 26, "everything seemed to be trembling" when she awoke and she saw double. This symptom lasted for six days and was most noticeable in the morning. The next day when she tried to get up she found she could not walk and she also had difficulty in speaking. The following morning she could not use her arms and she had trouble in swallowing. There was no pain or discomfort and no unusual sleepiness. The physician's examination at this time showed that pulse, temperature and respiration were normal. The uvula was edematous and covered with a profuse mucous exudate (negative for diphtheria bacilli). The pupils were equal and somewhat dilated; there was slight twitching of the eyelids. The superficial and deep reflexes were normal; muscular coordination was good and there was no paralysis. The tentative diagnosis was hysteria. She continued in about the same condition until her admission to the Psychopathic Hospital on December 12.

Examination.—On admission she felt very weak and uncomfortable, but had no actual pain. She was mentally clear and alert, quiet and cooperative. The neurologic examination at this time showed: pupils widely dilated, the right slightly the larger; both reacted in a limited degree to light and better on accommodation; partial bilateral ptosis; transient external strabismus of the left eye; facial movements were good with the exception of the orbicularis oris; marked difficulty in speaking and in protruding the tongue, which was, however, in the midline and showed no tremor; much difficulty in swallowing; mastication normal; hypersecretion of mucus in the throat. The patient could move the arms and legs but said they felt heavy; power was fairly good against resistance; finer movements of the fingers were poorly carried out. Tendon reflexes: biceps sluggish; triceps not obtained; patellar and Achilles normal. There was no Babinski sign. Some difficulty existed in urination. The chest and abdomen were negative.

Course.—The patient seemed to improve during her stay in the hospital. She was stronger and had less difficulty in talking and swallowing. She died very suddenly, December 19, while being given a bed bath. The duration of the illness was twenty-three days.

Case 3.—E. M. D., a girl aged 16, is especially interesting both from a psychiatric standpoint and because there is the possibility of two cases of epidemic encephalitis in the same family. The history was essentially negative up to the time of her last illness. A few months previously the patient's twin sister (L. D.) had been the victim of an assault and since had been depressed and worried over the matter. Early in March, 1922, she (L. D.) was taken with "grippe." Two days after the onset she became confused and violent. Attacks of great excitement alternated, at first, with quiet and a

return to normal mentality, later with stupor. She continued in the same condition until her death at home, April 16. The diagnosis made was dementia praecox. Our patient (E. M. D.) was deeply attached to her sister and had been much impressed by the assault. She was with her sister constantly during the first week of the latter's illness. March 20, she was seized with a convulsion and following this she was restless, sleepless, refused to eat or talk, and became violent when any effort was made to control her. Later she was in a stupor most of the time. She was admitted to the Psychopathic Hospital April 18.

Examination.—On admission the patient was restless and did not cooperate. At times she showed some flexibility and was negativistic. Physical examination revealed: much emaciation; marked diminution of tactile sensibility; pupils dilated but reacting to light. There was an Oppenheim sign on the left. Other neurologic tests were negative. The general examination was negative, except for a very rapid heart rate (132). The spinal fluid showed no globulin, normal albumin and a negative colloidal gold test. The temperature was 100.8 on admission, but was subnormal for two days before death. The patient vomited her tube feedings, became weaker, cyanotic, and died April 20, after an illness of one month. This case might easily have been interpreted from a psychogenic standpoint, the mental symptoms representing a hysterical repetition of her sister's illness but a consideration of all the factors makes the organic basis of the condition clear.

Cases 4 and 5 were examples of the myoclonic form of the disease.

Case 4.—J. L., a man, aged 38, a clerk with a past history that is unimportant, noticed in the latter part of December, 1920, numbness of the hands and stiffness of the fingers. About Jan. 4, 1921, he stopped work on account of headache; January 18, this was severe enough to cause him to go to bed. He was very irritable, talkative and restless at this time. The numbness in his hands persisted and about January 14, he had pains in his feet and legs. He was unable to sleep on account of the pain and headache. January 20 his temperature was 101. Twitchings of the trunk and extremities were first noted by the family on January 22. He was then sent to a general hospital, where he soon became confused and hallucinated. He was admitted to the Boston Psychopathic Hospital January 25. Here he was restless, confused and disoriented, pulled at the sheets, thinking it was clothing to put on, talked to his children at home, etc.

Examination.—Physical examination disclosed: the pupils reacted sluggishly to light; nystagmus of both eyes, increased on lateral or upward movement; weakness of the levator palpebrae and an inability to close the eyes completely; the deep reflexes were slightly increased, especially the knee jerks; there was no ankle clonus. The abdominal muscles showed shock-like contractions, occurring from twenty to thirty times a minute. These contractions extended occasionally into the muscles of the thighs, causing them to flex on the abdomen. The movements of the thighs and abdominal muscles were simultaneous. They were more marked on the right side and were accompanied by severe pain. The abdomen was slightly tender on pressure. On the two days before death the abdominal contractions decreased to ten or twelve a minute. There were also occasional twitchings of the arms and marked coarse tremors of the forearms and outstretched fingers, the latter increasing on voluntary movement.

The temperature gradually rose from 101.4 on admission to 104 on the day of death. The patient passed into coma and died February 1, after an illness of four weeks.

CASE 5.—A. H., a man, aged 31, a factory operative, with a past history that is unimportant, for two months complained that he felt run-down and could not work as well as formerly. About three weeks before admission to the Boston Psychopathic Hospital he was said to have had influenza, although he was in bed only one day. A week later, March 26, 1922, after doing some work around the yard of his home, he complained of pains in his arms. These grew so severe that they prevented sleep. He became delirious at night and twitchings in the arms were noticed. After a few days he was constantly delirious and complained of headache and pain in the neck and back. He was brought to the hospital April 5.

Examination.—Physical examination showed the pupils almost rigid to light but reacting on accommodation. There was a lateral nystagmus. The deep reflexes were present in the upper extremities; the right knee jerk was absent, the left diminished; the ankle jerks were normal. There was a tendency to a Babinski sign on the right. There were clonic contractions of the muscles of the upper part of the abdomen, occurring about twenty-five times a minute. These occurred in cycles of three to seven strong contractions, then a few weaker ones, followed by an interval of rest; then a repetition. The patient seemed to hold the muscles tense and in this way he could control the movements to a slight extent. There was no abdominal tenderness. There were also twitchings of muscle groups in the right arm and forearm and at times irregular jerkings of the legs.

The temperature varied from 100 to 101 for five days after admission. April 11, signs of pneumonia were apparent and the patient died April 12, after an illness of seventeen days.

HISTOLOGIC FINDINGS

Interest in the cerebral cortex in lethargic encephalitis has been subsidiary to that in the midbrain and lower centers, and consequently much less attention has been devoted to it. It has frequently been pronounced negative after examination of a few sections. Some writers have found neuronophagia, hemorrhages and slight perivascular exudate. Others, who have been interested primarily in the finer histology of the disease, have described the lesions without particular reference to their situation, although they mention certain of them as occurring in the cerebral cortex. Hassin [1] has emphasized the difference between toxic and infectious encephalitis, using lethargic encephalitis as an example of the latter type; he contrasts the productive lesions of the toxic with the infiltrative lesions of the infectious type of encephalitis. Buzzard [2] and Watson [3] have reported cases of lethargic encephalitis in which the maximum of the disease was found in the cortex, but their cases differed much from the ordinary form of the disease clinically,

1. Hassin, G. B.: The Contrast Between the Brain Lesions Produced by Lead and Other Inorganic Poisons and Those Caused by Epidemic Encephalitis, Arch. Neurol. & Psychiat. **6**:268 (Sept.) 1921.

2. Buzzard, E. F.: Lethargic Encephalitis, Lancet, **2**:835 (Dec. 21) 1918.

3. Watson, G. A.: Encephalitis Lethargica Involving Chiefly the Cerebral Cortex, J. Neurol. & Psychopath. **1**:34 (May) 1920.

by showing paralyses and aphasia, and pathologically, by having large areas of softening and hemorrhage; further, no mention is made of the lesions in the midbrain.

As no special study seems to have been made of the distribution of the cortical lesions in epidemic encephalitis, our attention was directed particularly to this point, to see whether, like the changes in general paralysis, they predominated in any special areas.

Definite changes were found in the cortex in all the brains. They were of the same general nature in all, though they differed in intensity and relative prominence in the different cases. Cell changes were slight in all except Case 1. They consisted in mild chromatolysis and occasional shrunken and diffusely staining cells with eccentric nuclei. The affected cells were scattered among normal ones in all layers. In Case 1, which was of the shortest duration (twelve days), the changes were marked and disintegrating cells were numerous.

Increased satellitosis was also present in all cases, though it varied considerably from case to case and section to section. It was most abundant, as is usual in the deeper layers of the cortex. Neuronophagia was evident only in Case 1, in which it was quite prominent. Neuroglia cells having large, light-staining nuclei and visible cytoplasm were numerous in Cases 1 and 5, especially around the nerve cells; they probably represented transitional stages to ameboid glia.

Perivascular infiltration was slight, but was found in all cases, though not in all areas. Usually one or two vessels to a section were surrounded by lymphocytes. Plasma cells were sometimes found in the tissue. Congestion of the cortical vessels was seen in all sections, and occasional hemorrhages into the perivascular spaces were present.

Considerable accumulations of degeneration products, presumably lipoid, occurred around the vessels in all cases. These stained green with cresyl violet and brownish with phosphotungstic acid hematoxylin, and were present chiefly around the veins in the white matter. The fat-content of the cortex was very moderate in all except Cases 2 and 3, in which there was a heavy deposit around the vessels and in the nerve cells in all regions. It is interesting to note in this connection that the mentality of the second patient was practically normal.

In regard to the topography of the above described lesions, no special distribution could be made out. They were scattered irregularly through the various cortical areas, and the occipital region was as often affected as the frontal or central. The congestion and the lipoid deposits were especially diffuse in their distribution.

The changes in the corpus striatum were of the same general nature—chromatolysis, satellitosis, slight perivascular exudate, and lipoid pigment. There was also some diffuse infiltration of the tissue and clumps of glia nuclei. The lesions were slight in all cases except Case 1, in which, as has been said, the changes in the cortex were more marked and more acute than in the other brains.

In Case 1 the putamen showed a rather unusual degeneration product, which appeared as homogeneous globules of various sizes, that stained an intense uniform dark blue with cresyl violet and hematoxylin. They were found chiefly among the fiber bundles as scattered clusters or as beading along the vessels. They were present nowhere else in the nervous system nor in any other case. Similar bodies have been described in the basal ganglions in epi-

demic encephalitis by Calhoun;[4] also by Faworsky[5] among the degeneration products in the cord in tabes. Fulci[6] has described, in a case of anthrax encephalitis, what seems to be a similar material, but in his case it occurred in large masses that filled the perivascular spaces. The nature of this substance is unknown, but it apparently belongs among the basophilic degeneration products of Alzheimer.

The thalamus and hypothalamus presented the same lesions as the corpus striatum. In general they were moderate in degree, and in Case 2 they were almost lacking. Both thalamus and hypothalamus were equally affected.

The midbrain was in all instances the site of the usual intense lesions. It may be mentioned that the red nuclei, the corpora quadrigemina and the geniculate bodies shared in the general marked involvement.

The changes in the cerebellum were slight and consisted of foci of exudate in the pia, occasional cloudy swelling of the Purkinje cells, small collections of lymphocytes in the white matter, and very rarely a little perivascular exudate.

The lesions in the brain stem and cord are considered in the discussion of the myoclonic cases, as it was in these regions that differences were found between the myoclonic cases and the others.

The optic nerves were examined in all cases. In Case 1 there was fresh hemorrhage into the pial sheath, and in Case 5 hyaloid droplets were present. The others were normal. There were small collections of lymphocytes in the olfactory tract in two instances.

Exudate was frequently present among the roots of the other cranial nerves at their points of origin, but it did not extend far outward. Burrows[7] has particularly emphasized the involvement of the cranial nerves, but the changes found in our cases were not as great as he reports.

Nerve bundles from the cauda equina were normal in all cases except Case 5, in which there was slight focal perivascular infiltration.

The gasserian ganglions showed changes in Cases 1, 3 and 5. The cells stained diffusely or were shrunken and pyknotic; the vessels were congested and there was considerable diffuse lymphocytic infiltration, which in one instance extended out among the nerve bundles. Neuronophagia and hemorrhage were absent. The writer has frequently found infiltration of the gasserian ganglions in various kinds of cases, not only when there was an inflammatory lesion at the base, as meningitis, but also in syphilis of the nervous system, and in noninflammatory conditions such as dementia praecox, alcoholic dementia, etc.

Specimens of the posterior root ganglions were examined in three cases, including one of the myoclonic. They were all normal.

No changes were observed in the ependyma of any of the ventricles or of the aqueduct.

The choroid plexus was examined in three cases. Two presented no abnormality but the plexus of the fourth ventricle of Case 5 showed a large

---

4. Calhoun, H.: Histopathology of the Brain and Spinal Cord in a Case Presenting a Post-Influenzal Lethargic Encephalitic Syndrome, Arch. Neurol. & Psychiat. **3**:1 (Jan.) 1920.

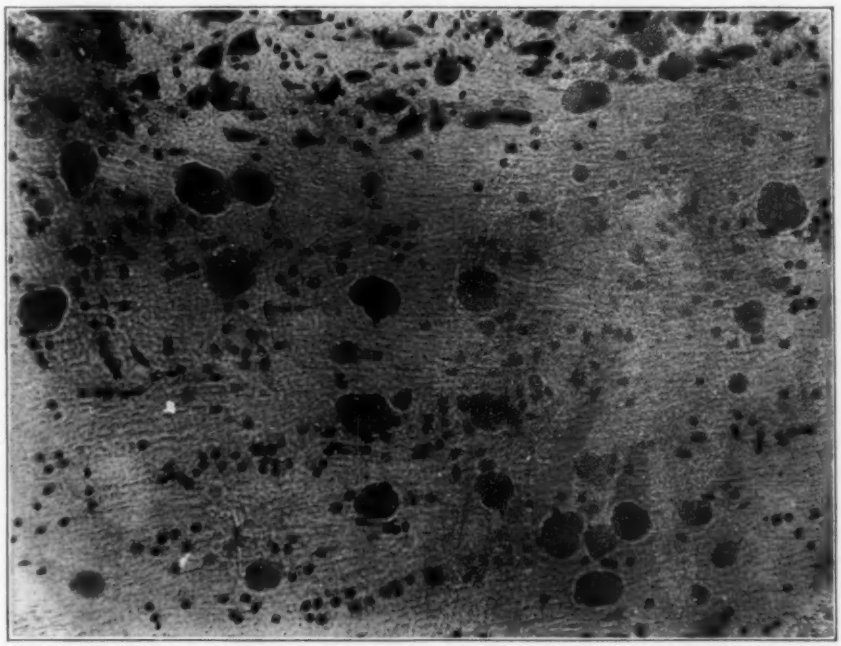
5. Faworsky, A.: Histol. u. histopathol. Arb. über die Grosshirnrinde, Nissl and Alzheimer **6**:74, Part 1, 1913.

6. Fulci, F: Histol. u. histopathol. Arb. über die Grosshirnrinde, Nissl and Alzheimer **6**:160, Part 2, 1913.

7. Burrows, M. T.: Neuritis of the Cranial Nerves in Lethargic Encephalitis, etc., Arch. Int. Med. **26**:477 (Oct.) 1920.

vessel containing a white thrombus. The surrounding plexus contained considerable fresh hemorrhage.

A noteworthy finding was the presence in Case 2 of an enormous amount of a degeneration product resembling colloid. This appeared as sharply outlined refractile droplets of various sizes (shown in the accompanying figure). The larger masses frequently had convoluted edges. They stained bright pink with thionin, extremely pale pink with cresyl violet and phosphotungstic acid hematoxylin and were unstained by alum hematoxylin, eosin and scharlach R. With Best's method for glycogen they took a pale mauve tint. They were most abundant in the midbrain and upper pons, where both gray and white matter were studded with them. They were abundant also throughout the rest of the pons and in the medulla, numerous in the corpus striatum and thalamus, and



Midbrain, (Case 2), showing the large amount of colloid material.

present in small numbers in the various cortical areas. They were also scattered through the white matter of the cervical cord, but not below this level. Degeneration products, similar in a general way to the above, have been described in epidemic encephalitis by Calhoun,[4] Boyd[8] and Harris.[9] Watson[3] also has described and pictured, in the neighborhood of vessels, rarefied sieve-like areas which frequently contain an amorphous deposit staining deep mauve with toluidin blue, lighter mauve with Van Gieson's stain and brownish orange with pyronin methyl green. These substances described by the various writers vary somewhat in staining reactions, judging from the accounts given,

8. Boyd, W.: Acute Epidemic Encephalitis, New York: Paul B. Hoeber, 1921, p. 189.

9. Harris, A.: A Case of Epidemic Encephalitis, Am. J. Psychiat. **1**:679 (April) 1922.

but they all seem to belong to the ill-defined group of so-called "colloid" of the central nervous system. Boyd surmises that they are derived from neuroglial degeneration.

The changes described above in the cortex, basal ganglions and midbrain were common to all the cases. In the study of the brain stem and cord it was found that both the myoclonic cases presented a similar picture with minor variations and differed from the others in two respects; first, in the greater intensity of the process in the pons and upper medulla; and second, in the presence of lesions in the cord.

In regard to the pons and medulla, the myoclonic cases exceeded the others in the degree and extent of the exudate rather than in the nerve cell lesions. The latter were prominent in all the cases and consisted in scattered degenerating cells, numerous cells showing chromatolysis, and considerable satellitosis. The affected cells were most numerous in the dorsal portion, where they were scattered through all nuclei. They were not confined to the dorsal side, however, but were present to some extent also in the pontine and olivary nuclei. On the whole, it is impossible to say that one nucleus in the dorsal region was more involved than another, except that the locus ceruleus always showed marked lesions, and abnormal cells were always numerous near the raphe and in the formatio reticularis. Perivascular exudate was abundant and widespread throughout the pons and medulla in the myoclonic cases. In Case 4 it was heavier in the upper pons than in the midbrain. There was also a diffuse increase of nuclei throughout the tissue and proliferation of the vascular endothelium. The pia was infiltrated throughout the length of the medulla. The intensity of the lesions, both cellular and infiltrative, diminished as the lower levels of the medulla were reached. In the non-myoclonic cases exudate was scanty and was absent in many sections.

Involvement of the cord was evidenced macroscopically in Case 5 by fresh adhesions between the dura and pia in the cervical region and by the presence of bloody fluid in the thecal sac. Both myoclonic cases showed marked perivascular exudate in the lower cervical and a lesser amount in the lumbar cord. In the cervical region the exudate appeared both in the gray matter of the anterior and posterior horns and in the white matter; in the lumbar cord only in the anterior horns. Exudate was limited to the cervical and lumbar enlargements but the nerve cell changes were more widely distributed. They consisted chiefly in cells showing the axonal reaction, which were found in the cervical and lumbar and also in the dorsal regions. The dorsal cord was the least involved in both instances, showing nothing beyond a few cells giving the axonal reaction. This is particularly noteworthy, as the muscles most concerned in the myoclonic movements were the recti abdominis, which are innervated from the fifth to the twelfth dorsal segments.

The pia in the cervical and lumbar regions was mildly infiltrated. Both anterior and posterior roots, as they appeared in sections, were normal.

The non-myoclonic cases showed no exudate. The gray matter was congested in all cases and in Case 3 there were two extensive areas of hemorrhage into the perivascular spaces of the posterior horn of the fourth cervical segment. Minor cell changes were present in the cervical region of Case 1.

In summary then, the distribution of the lesions in the myoclonic cases was as follows: diffuse and mild in the cortex, corpus striatum and thalamus; of the usual intensity in the midbrain; very marked also in the pons; decreasing

gradually throughout the medulla; marked again in the cervical region of the cord; mild or absent in the dorsal region; and slight in the lumbar region of the cord.

In some of the reported necropsies on cases of epidemic encephalitis showing movements of the myoclonic type, the lesions have been described as unusually severe in the pons and medulla; and when the cord has been examined, it has generally been found involved.

In Winkelman and Weisenburg's [10] case there were twitchings all over the body, and pains which were sharply limited to the head, upper limbs, chest, and abdomen to the level of the umbilicus. "The pathologic findings were those which are usually present, but careful study of the cord demonstrated that there was a lessening of the pathologic process as the lower levels of the cord were reached, and that the cord below the lower thoracic level was altogether normal."

In Hassin's [11] case there was enormous infiltration in the pons, midbrain and locus niger, both perivascular and invading the parenchyma. The cord was not removed.

Gerlach [12] reports two cases, both of the lethargic type, that showed muscular twitchings; in the first patient these involved the entire body; in the second, the abdominal muscles and one hand. In both cases the lower dorsal and upper lumbar regions were involved, the other parts of the cord only slightly. There were marked changes in the medulla.

Häuptli [13] gives short accounts of three cases in which the cord showed marked infiltration. In the first there was pain in the abdomen and left arm, twitchings in the latter, hyperalgesia of the left leg, increased knee jerks, and ankle clonus. The cervical cord was diffusely infiltrated. In the dorsal cord exudate was less abundant and was focal. The second case showed, at the onset, pain in the right shoulder, abdominal muscles and right leg, and later, clonic contractions in these groups, also hiccough. The maximum of the lesion was not in the midbrain but in the medulla, and it continued with special severity throughout the entire cord. In the third case there were myoclonic twitchings in the arms. The lesions were less intense in the midbrain and medulla than in the cerebellum and cervical cord.

---

10. Winkelman and Weisenburg: A Case of Myoclonic Lethargic Encephalitis Presenting Unusual Pathologic Manifestations, Arch. Neurol. & Psychiat. **5**:224 (Feb.) 1921.

11. Hassin, G. B.: Two Cases of Atypical Epidemic Encephalitis, J. Nerv. & Ment. Dis. **53**:217 (March) 1921.

12. Gerlach, W.: Ueber Rückenmarksveränderung bei Encephalitis lethargica, Berlin klin. Wchnschr. **57**:585 (June 21) 1920.

13. Häuptli, O.: Zur Histologie der Poliomyelitis acuta und der Encephalitis epidemica, Deutsch. Zeitschr. f. Nervenh. **71**:1 (May) 1920.

Ducamp and Carrieu [14] report a case of epidemic hiccough with myoclonia. The lesions were especially localized in the pons, medulla and cervical cord. The lesions in the peduncles were less intense than usual. In the medulla, degeneration was found principally in the vago-spinal nucleus. The dorsal and lumbar cord were only slightly affected.

Roubier [15] reports three myoclonic cases in which the lesions were most marked in the pons and locus niger.

Valente and David,[16] in three necropsies on the pure myoclonic form of Sicard, found diffuse, very grave lesions of the cord, striking, with evident election, the segments which corresponded to the most marked pains and myoclonic movements. In one case the authors found no lesions above the medulla; in the two others there were pontine and midbrain lesions, with normal basal ganglions and cortex. The lesion was a total acute poliomyelitis, striking the gray matter in its entirety.

In Bostroem's [17] and Bradley's [18] cases the findings were different from those given above. In Bostroem's two cases the most affected areas were the floor of the fourth ventricle, the brachium conjunctivum and the lenticular nucleus, particularly the median part of the globus pallidus. No mention is made of the cord. In Bradley's case the lesions were in the midbrain and pons. The medulla showed little abnormality and the cord none.

Our own results are indecisive as far as locating any particular lesion responsible for the myoclonia is concerned, and most probably nothing so definite and elementary is to be expected. Although there is a tendency for myoclonic cases to show lesions in the cord, it does not seem as if these could be directly connected with the movements, for at least one necropsy has been reported in which the cord was negative; on the other hand, there have been cases described in which the cord was much involved but no myoclonia was present. Moreover, the distribution of the cord lesions, in our cases at least, is not what one would expect, for although the recti abdominis were the muscles most affected, the dorsal cord, from which they are innervated, is the least involved portion. The variation of the pathologic findings in the reported cases shows the complex and unsettled state of the question.

---

14. Ducamp and Carrieu: Hoquet epidémique avec mouvements myocloniques généralisés, Bull. Acad. d. méd. de Paris **86**:249, 1921.

15. Roubier: Lyon Méd. **129**:452, 1920.

16. Valente and David: Sur la poliomyélite myoclonique, Compt. rend. Soc. biol. de Paris **83**:1390 (Oct. 25) 1920.

17. Bostroem, A.: Ungewöhnliche Formen der epidemischen Enzephalitis unter besonderer Berücksichtigung hyperkinetischer Erscheinungen, Deutsch. Zeitschr. f. Nervenh. **68**: and **69**:64 (Jan.) 1921.

18. Bradley, E.: A Case of Myoclonic Epidemic Encephalitis, Brit. M. J. **1**:891 (June 18) 1921.

The prevailing opinion from clinical evidence in regard to the site of the lesion responsible for myoclonic movements is that it is probably an irritative lesion of the lower motor neurone. The lancinating pains which are characteristic of the myoclonic form have suggested a participation of the posterior root ganglions in the pathologic process and also that they may be the point of entrance of the virus. The former idea has not been borne out by the one of our myoclonic cases in which the ganglions were examined. Both Mingazzini [20] and Walshe [21] consider the myoclonia due to infiltration of the anterior roots. As has been said, the anterior roots in all our cases were normal.

The findings in the organs of the trunk were as follows: all the organs showed congestion, which was most striking in the spleen. In Cases 2, 3 and 4 the lungs contained areas of fresh hemorrhage, and in Cases 3 and 4 there was an early bronchopneumonia. The intestinal tract, in Cases 1 and 4, was the site of definite lesions. In the former the intestine, especially the ileum, was injected macroscopically and in the lower ileum the mucosa was thickened and covered with fluid blood. The mucosa of the cecum was thickened and presented numerous ulcers, which had red edges and yellow bases, beneath which was hemorrhage showing through on the peritoneal coat. The cecal lymph nodes were enlarged and purple. Microscopically the thickened areas showed a superficial necrosis, and beneath the surface were many large mononuclear phagocytes. The lymphoid tissue was increased in amount and active. The vessels were injected and those in the muscular coat were lightly infiltrated with lymphocytes. In Case 4 the gastric mucosa showed petechial hemorrhages and the upper part of the duodenum and lower portion of the ileum were much injected. The congestion and petechial hemorrhages are similar to those frequently found in other organs in epidemic encephalitis, but the changes in Case 1 are unusual. The significance of this lesion and whether it has any relation whatever to the disease is problematic. There is no mention of intestinal symptoms at any period of the disease. Lymphoid hyperplasia, either in the intestine or other organs, is not a part of the pathology of epidemic encephalitis as it is of poliomyelitis, nor are intestinal symptoms frequent, although Dana [22] mentions their occurrence at the

20. Mingazzini, G.: Klinischer und anatomisch-pathologischer Beitrag zum Studium der Encephalitis epidemica, Zeitschr. f. Neurol. u. Psychiat. **63**:199, 1921.

21. Walshe, F. M. R.: On the Symptom-Complexes of Lethargic Encephalitis with Special Reference to Involuntary Muscular Contractions, Brain **43**:197 (Nov.) 1920.

22. Dana, C. L.: Acute Epidemic Encephalitis, New York: Paul B. Hoeber, 1921, p. 133.

beginning and Barker [23] during the course of the disease. There seems to be nothing in the literature on lesions of the intestinal tract in epidemic encephalitis aside from an occasional description of congestion found at necropsy.

The pituitary gland was normal in all cases except for congestion. In three instances there seemed to be an unusually large proportion of basophil cells in the pars anterior in sections taken directly through the center of the gland, but the distribution of basophils and acidophils is normally so irregular that it is difficult to judge of their proportions.

SUMMARY

Systematic examination of various parts of the central nervous system in epidemic encephalitis has shown that although centered in the midbrain, the disease process affects all parts of the cerebrospinal axis. Practically no region is entirely normal. Except in the midbrain the lesions are scattered and focal, the affected cells and vessels being found among others which appear quite normal.

In the cerebral cortex the lesions are irregularly and diffusely distributed and no one region is more affected than another. The same cortical changes are found in patients who have shown very different mental symptoms.

The myoclonic cases were distinguished from the others by the greater severity of the process in the pons and medulla and by involvement of the cord. The parts affected were the cervical and to a lesser extent the lumbar regions.[24]

Various degeneration products in the central nervous system and the presence of intestinal lesions are described.

Boston Psychopathic Hospital.

---

23. Barker, L. F.: Acute Epidemic Encephalitis, New York: Paul B. Hoeber, 1921, p. 133.

24. Since this article was written another case of epidemic encephalitis with myoclonia of the abdominal muscles has come to necropsy at the Boston Psychopathic Hospital. The sections show an enormous infiltration in the midbrain and the dorsal portions of the pons and medulla. In the cervical and thoracic regions of the cord there is also a heavy perivascular infiltration in the posterior horns, and around occasional vessels in the white matter. The lumbar cord is not affected.

STABILIZING BRAIN TISSUE DURING FIXATION

GEORGE S. STEVENSON, M.D.
Assistant in Neuropathology, Psychiatric Institute.
WARD'S ISLAND, N. Y.

In the fixation of tissue it is the aim to hold the structures as nearly as possible in the form they had during life, or at the moment when the fixing reagent acted. Exceptionally, an artefact may have a differentiating value; with this we are not concerned here. But when conditions exist which can be recognized as artefact, it is desirable to eliminate them, or to evaluate them.

At present 10 per cent. liquor formaldehydi, U. S. P., is the most extensively used fixative for whole brains. In dealing with unknown brain tissue so fixed, conclusions regarding deviations from the normal picture are based on our experience with formaldehyd fixed tissue. Certain conditions due to fixation rather than to antemortem influences are eliminated; for example, certain clouding of the pia-arachnoid is expected to appear after exposure to formaldehyd. In finer histologic work, detectable change in structure, due to fixation, is apt to vary in intensity and must be considered objectionable, and should be eliminated if possible. Such changes may obscure or be confused with actual antemortem changes.

Two conditions found after the fixation of brains in 10 per cent. liquor formaldehydi, U. S. P., prompted a study of some of the changes attending such fixation, and a search for methods of preventing those that were objectionable. Two conditions that we considered undesirable, are apt to occur in brain tissue whether pathologic changes are present or not. They were especially brought to our attention since they appeared in tissues intended for use as normal controls.

The first of these two changes was the swelling and change of weight that took place after the time of necropsy and during fixation. It is fairly well known that a brain immersed in 10 per cent. liquor formaldehydi at necropsy gains considerably in weight for a while, increasing usually 100 or more grams. It then falls again, but usually does not return to its original weight. If after fixation such a brain be transferred to a solution of 10 per cent. liquor formaldehydi in physiologic sodium chlorid solution, it falls below its original weight. The following observation is an example of such a change:

A brain at necropsy (Nov. 13, 1920) weighed 1,698 gm. and was placed in 5 per cent. formaldehyd solution. Two weeks later, Nov. 29, 1920, the brain

weighed 1,805 gm., a gain of 107 gm., and was then transferred to 4 per cent. formaldehyd in physiologic sodium chlorid solution. Subsequent weights in grams were:

Dec. 13, 1920	1,745
Dec. 27, 1920	1,705
Jan. 19, 1921	1,675
April 15, 1921	1,651
May 18, 1921	1,635
Aug. 3, 1921	1,620

We are faced with the problem of finding a method of fixing tissues that would reduce these volume alterations.

The second change consisted of the so-called shrinkage alteration of the nerve cells of the second and part of the third layer, or of those even deeper. While not a constant change, it was so frequent as to excite doubt as to its pathologic significance. Aside from its own significance, it is a change that would be apt to obscure a slight, but truly pathologic, alteration in these cells. The claim has been made by Hoch,[1] that such changes sometimes arise from treatment during fixation and other technical procedures. He remarked in 1898 that "we . . . (cannot) . . . more than indicate the difficult problem which arises from the combination of this with other changes." He was able to duplicate these changes by treating fresh rabbit brain tissue with distilled water. Dunlap has called attention to the fact that the cortex deep in the sulci, where protected from rapid fixation, was usually free from this alteration. He suggested that some action on the unprotected surface probably had a bearing on this change. Whether the practice of washing the surface blood from a brain prior to fixation has anything to do with this change is a question. At any rate it was considered desirable to eliminate it if possible.

From our experience with the behavior of brains after treatment with formaldehyd and with formaldehyd and sodium chlorid, we concluded that alteration of the salt content of the fixing medium might be taken as the basis of procedures designed to reduce these undesirable changes. It had previously been shown by Bauer and Ames [2] that the tissue reacts very differently to different solutions, depending on the particular salt present. This indicated that selection would be advisable in the salt used for the solution. However, we thought that control of the concentration of the solution would do much toward stabilizing the brain during fixation.

---

1. Hoch, August: Nerve Cell Changes in Somatic Diseases, Am. J. Insan. **55**:231, 1898.

2. Bauer and Ames: Contributions from the Third Division of the Neurological Institute, New York: June, 1914.

In attacking the problem, the plan followed was first to experiment with small bits of tissue weighing from 0.2 to 1.0 gm. These could be obtained easily, handled easily, and their reactions to the reagents determined quickly. A brief survey with various strengths of formaldehyd was first made. It was

TABLE 1.—*Fixation Experiments with 10 per cent. Liquor Formaldehydi and Varying Concentrations of Sodium or Copper Sulphate* *

No	Disease	Salt	Percentage Concentration	Changes Made
1	General paralysis	Sodium sulphate	0.9 0.5 0.0	Two changes Twelfth day Seventeenth day
2	Arteriosclerosis	Sodium sulphate	0.9 0.5 0.0	Two changes Seventh day Tenth day
3	General paralysis	Sodium sulphate	0.9 0.5 0.0	Two changes Eighth day Twelfth day
4	Large cerebellar softening	Sodium sulphate	0.9 0.5 0.0	One change Seventh day Fifteenth day
5	General paralysis	Sodium sulphate	0.9 0.5 0.0	32.54 and 30 c.c. of liquor formaldehydi added on second, fourth and fifth days respectively for percentage reduction determined by analysis Seventh day Tenth day
11	Arteriosclerosis	Copper sulphate	0.7 0.6 0.5 0.5 0.0	 Third day Fifth day Seventh day Eleventh day
12	Dementia praecox	Copper sulphate	0.7 0.6 0.5 0.3 0.0	 Second day Fourth day Eighth day Tenth day
13	General paralysis	Copper sulphate	0.7 0.5 0.3 0.0	 Fourth day Sixth day Eighth day
14	General paralysis	Copper sulphate	0.8 0.5 0.0	24 and 30 c.c. of liquor formaldehydi added on second and third days respectively for percentage reduction determined by analysis Fourth day Seventh day
21	Dementia praecox	Control	...	Changed twice

* A standard of 3,000 c.c. was used as a fixing solution. The weights were taken after the brain had stood for twenty minutes, base down on a moist towel.

concluded that 10 per cent. liquor formaldehydi, U. S. P., as usually used was most satisfactory. Greater concentrations penetrated less rapidly, lesser concentrations caused too much mass increase. Alcohol combined with 10 per cent. liquor formaldehydi was tried, but no mixture of these was found which reduced the mass changes. In fact the rebound mass shrinkage was increased.

A number of salts in various concentrations were tried in combination with 10 per cent. liquor formaldehydi. These included magnesium sulphate, sodium

chlorid, sodium sulphate, ammonium chlorid and copper sulphate. Of these salts sodium sulphate and copper sulphate were most satisfactory, as they restrained swelling in lower concentrations than the others. The molecular content of the solutions of different salts seemed to bear no direct relation to their effects. With the same salt the effect, in restraining swelling, varied directly with the concentration. It was accordingly determined to limit further studies to these two salts.

It was next necessary to determine in what concentrations copper sulphate and sodium sulphate were most satisfactory. For this purpose a series of graded concentrations was made in 10 per cent. liquor formaldehydi; small bits of tissue

TABLE 2.—*Changes in Weight of Brains Described in Table 1.*

Brain	Necropsy Weight	Maximum Weight	Minimum Weight	Fourth Week Weight	Weight of Portions* Removed for Examination (Estimated)
1	1,062	1,062	1,035 (14th day)	1,052	9 gm.
2	1,119	1,040 (7th day)	1,119 (35th day)	1,124	9 gm.
3	1,202	1,210 (5th day)	1,195 (20th day)	1,196	6 gm.
4	1,310	1,310 (1st day)	1,229 (12th day)	1,246	6 gm.
5	1,227	1,230 (11th day)	1,216 (4th day)	1,229†	6 gm.
11	1,228	1,245 (3d day)	1,192 (22d day)	1,195	6 gm.
12	1,198	1,222 (3d day)	1,172 (35th day)	1,174	5 gm.
13	1,220	1,259 (6th day)	1,220 (1st day)	1,240	6 gm.
14	1,319	1,340 (4th day)	1,319 (1st day)	1,330†	6 gm.
21	1,322	1,565 (5th day)	1,322 (1st day)	1,535	6 gm.

* The pieces removed for examination were taken before the fourth week, consequently their weights must be added to the weight of the fourth week for proper comparison.
† Taken at the end of two weeks.

TABLE 3.—*Maximum Changes in Weight of Brains*

Fixative	Brain	Maximum Gain or Loss in Gm.
Graded sodium sulphate in 10 per cent. liquor formaldehydi.....	1	−27
	2	−21
	3	+ 8
	4	−81
	5	−11
Graded copper sulphate in 10 per cent. liquor formaldehydi.....	11	−36
	12	+29
	13	+39
	14	+21
Ten per cent. liquor formaldehydi.....	21	+243

were fixed in these solutions and weighed daily during fixation, and for several weeks afterward. It was found that from 0.8 per cent. to 0.9 per cent. sodium sulphate[3] and 0.7 per cent. copper sulphate held the tissue most constant during its fixation period. On following this tissue over a longer period, however, it was found to steadily decrease in weight if left in the original fixative of 0.7 per cent. copper sulphate or 0.9 per cent. sodium sulphate in 10 per cent. liquor formaldehydi. Some pieces decreased as much as 35 per cent. of their original weight.

Having determined that for small pieces 0.9 per cent. sodium sulphate and 0.7 per cent. copper sulphate in 10 per cent. liquor formaldehydi was most

3. All percentage calculations were made on the basis of the anhydrous salt.

promising, it remained to try out these solutions in the fixation of whole brains, and to study brains, so fixed, microscopically for artefacts. It was foreseen that a single solution would not suffice to keep the brain weight constant, as the tissue behaves differently at different stages of fixation. It was thought that by reducing the salt concentration in steps, the brain might eventually be put in 10 per cent. liquor formaldehydi as its permanent preservative. Just when these changes would be necessary could be told only by trial with whole brains as the use of small pieces proved entirely unsatisfactory for this purpose. Consequently a series of brains was fixed in varying concentrations as shown in Table 1. In Table 2 are given the important data concerning changes in the weights of these brains.

DISCUSSION OF RESULTS

Examination of the tables of results shows that there is the same tendency toward swelling that appears in simple formaldehyd fixation, but in diminished degree. By the end of the first week with sodium sulphate solutions, and a little earlier with copper sulphate solutions, this swelling ceases and shrinkage begins. Weakening of the salt concentration at these times prevents this shrinkage to a large extent. While more gradual steps were made with some brains, it seems sufficient, with either salt to reduce first to a 0.5 per cent. solution and then to liquor formaldehydi 10 per cent. without salt. On the whole the sodium sulphate solutions stabilize the brains better than copper sulphate, but the copper solutions allow more rapid fixation.

Aside from the results of fixation, brains vary much as regards swelling and shrinkage, probably as the result of such factors as age, type of disease and the time that elapsed between death and the beginning of fixation. By the ordinary fixation method, the increase in weight of five brains observed during the period of swelling varied between 57 and 212 gm. In view of this individual variation, the following procedures are suggested as optimum stabilizing methods for all brains, rather than as perfect methods for any one brain:

1. Fix in 3,000 c.c. of sodium sulphate, 0.9 per cent.; liquor formaldehydi, U. S. P., 10 per cent. for 7 days. Then 3,000 c.c. of sodium sulphate, 0.5 per cent.; liquor formaldehydi 10 per cent.; for 3 to 5 days. Preserve in 3,000 c.c. of liquor formaldehydi 10 per cent.

2. Fix in 3,000 c.c. of copper sulphate, 0.8 per cent.; liquor formaldehydi, 10 per cent.; for 4 to 5 days. Then 3,000 c.c. of copper sulphate 0.5 per cent.; liquor formaldehydi 10 per cent.; for 2 to 3 days. Preserve, after short washing, in liquor formaldehydi 10 per cent.

In Method 1, two changes to fresh solutions should be made during the stage of 0.9 per cent. sodium sulphate. As an alternative, 30 c.c. liquor formaldehydi may be added on each of the second, third, fourth and fifth days.

In Method 2, 30 c.c. of liquor formaldehydi should be added on each of the second and third days.

From the standpoint of the gross appearance of the brain, the use of Method 1 leaves the brain with the same color as after simple formaldehyd fixation. Method 2, however, changes the color somewhat although the color washes out to some extent in the last solution. This method also makes the tissue firmer than does the first method.

Four stains, toluidin blue, hematoxylin, eosin and silver stains were tried on tissues fixed by these methods and were found to work satisfactorily.

The microscopic examination of the tissue for artefacts revealed an increase beyond the usual size of the perivascular and pericellular spaces. This feature was most prominent in the second layer, but occurred also deeper. While this is, of course, serious, it is probably dependent in part on the nature of the cases, i. e. the organic disease; for one case of dementia praecox was practically free from it. While the ordinary type of shrinkage alteration, the so-called sclerotic cell, was very infrequent, in two brains the closely related condition of crumbly or fragmented cytoplasm was present. In general, the nuclear detail was very good and the Nissl bodies were often demonstrable in the cells of the cortex of the frontal lobe.

In order to protect the surface of the cortex from the direct action of the formaldehyd, an area on Brain 4, was covered with glue. The glue became fixed and adherent to the surface. Pieces were then cut including this area and the adjacent uncovered cortex, and microscopic examination of these was made. Beneath the protected surface the cortex was a little paler than in the adjacent uncovered part. It resembled the sulcal cortex in this respect. A few dark shrunken cells were found in both areas.

CONCLUSIONS

1. It is desirable to stabilize brain tissue during the period of fixation.

2. Solutions of various salts affect brain tissue differently, independent of their concentration.

3. Sodium sulphate and copper sulphate have a strong restraining action on the swelling of brains during formaldehyd fixation.

4. Individual brains vary in their response to the same solution, hence a perfect fixing solution cannot be obtained.

5. One solution is inadequate for the whole period of fixation. Reduction of salt content as fixation proceeds is better.

6. The described methods are an improvement over simple liquor formaldehydi, 10 per cent., in that they tend to stabilize the tissue during fixation.

IMPLANTATION OF BACILLUS ACIDOPHILUS IN PERSONS WITH PSYCHOSES*

LOUIS A. JULIANELLE, PH.D.
AND
FRANKLIN G. EBAUGH, M.D.
PHILADELPHIA

HISTORICAL

In 1912, Berthelot and Bertrand [1] described an organism which they isolated from human stools and called *Bacillus aminophilus*. Among other characteristics, it showed a marked ability to decarboxylate and deaminize amino-acids. Also, it was capable of producing histamine from histidine. Holmes and Rettinger [2] alleged that dementia praecox was caused by the production of histamine by this organism in the intestinal tract.

From a study of the frequency of *B. aminophilus* in the stools of patients with dementia praecox, Jones [3] was unable to support the assumption of Holmes and Rettinger. He found: that this organism was present only in the proportion of from 1 to 20,000 to 1 to 100,000 of living bacteria; that it was found with a frequency usual in normal persons; that the histamine present was produced by other intestinal bacteria; that *B. aminophilus* did not utilize the carboxyl radical (COOH) of histidine if other sources of carbon were available; and that histamine (quoting Bayliss and Starling) is present in the normal intestinal mucosa. In short, Jones gives the impression that *B. aminophilus* bears no relationship to dementia praecox.

More recently, Cotton [4] has associated psychoses with focal infections, which begin at the teeth, and then spread to the stomach, duodenum and lower intestinal tract. Because of the frequency in such cases of the "Connellan-King diplococcus" and the colon bacillus, he suggests a causal relationship to the psychoses. In cases of lower

---

* From the bacteriologic laboratory and neuropsychiatric department of the Philadelphia General Hospital.

* Presented before the Philadelphia Pathological Society Nov. 23, 1922.

* Since this paper was submitted for publication, Kopeloff and Cheney have reported similar findings regarding the therapeutic effect of acidophilus milk, J. A. M. A. **69**:609 (Aug. 19) 1922.

1. Berthelot and Bertrand: Compt. rend. Acad. d. Sci. **154**:1826, 1912.

2. Holmes, Bayard, and Rettinger, J,: Lancet-Clinic **116**:145, 1916; Chicago Med. Rec. **38**:60, 1916.

3. Jones, H. M.: J. Infect. Dis. **22**:125, 1918.

4. Cotton, H. A.: New York M. J. **109**:397 and 454, 1919; The Defective, Delinquent and Insane, Princeton, N. J.: Princeton University Press, 1921.

intestinal infection he goes so far as to advocate colectomy, when this is necessary to eradicate the focus. He publishes reports of successful results following this procedure.

Metchnikoff introduced the ingestion of cultures of *Bacillus bulgaricus* in milk to eliminate intestinal putrefaction, on the assumption that the organisms and the acid of the milk cultures would be inimical to the putrefactive bacteria. Since then, however, it has been found that *B. bulgaricus* does not grow well in the intestine. This has led to other investigations of the possibility for elimination of intestinal putrefaction by changing the intestinal flora.

Rettger and Horton [5] and later Hull and Rettger [6] were able to change the intestinal flora of white rats from the ordinary type to an Acidophilic type by feeding milk or lactose. Torrey [7] found this to be possible also in typhoid fever in man. Finally, Rettger and Cheplin,[8] showed that it is possible to transform the intestinal flora of man in the majority of cases by the administration of broth cultures of *B. acidophilus*. In more refractory cases, the cultures were supplemented by the addition of lactose to the diet. Later, they introduced milk cultures of *B. acidophilus* and showed that then the *B. acidophilus* content rose to 85 to 95 per cent of the cultivatable bacteria, and that this bacillus was able to proliferate in the intestinal tract.

In planning our work, we had in mind these points: if dementia praecox is caused by intestinal stasis and subsequent putrefaction, the implantation of *B. acidophilus* might tend to remove the putrefaction, and possibly, therefore, to alleviate the psychosis.

EXPERIMENTAL

In our series of experiments, ten patients were studied; four were carried throughout the study; the others were dropped from time to time as indicated in case histories. A normal person was used as a control for the rapidity, degree and permanency of *B. acidophilus* implantation.

Analyses of stools were made for one week prior to feeding with acidophilus milk to show: (1) the character of the stools; and (2) the presence or absence of acidophilic organisms. With the inception of the feeding, examinations of the stools were made daily until implantation was established: then the schedule was changed to two examinations per week to determine whether the implantations were being sustained.

---

5. Rettger, L. F., and Horton: Centralbl. f. Bakteriol. **73**:362, 1914.
6. Hull, T. G., and Rettger, L. F.: J. Bacteriol. **2**:47, 1917.
7. Torrey: J. Infect. Dis. **16**:72, 1915.
8. Rettger, L. F., and Cheplin, H. A.: Treatise on the Transformation of the Intestinal Flora, with Special Reference to the Implantation of Bacillus acidophilus, New Haven, Conn.: Yale University Press, 1921.

The analyses consisted of: (1) staining smears by the gram method; (2) culturing fecal suspensions (in physiologic saline solution) on plates for numerical estimation of the *B. acidophilus* colonies; (3) culturing in litmus milk; and (4) culturing anaerobes in dextrose broth with the caustic soda-pyrogallic acid method.

The milk was prepared in bulk by heating in the autoclave at 5 pounds pressure for one hour. After cooling to room temperature, inoculations were made of about 2 c.c. of a twenty-four hour culture of *B. acidophilus*[9] to each liter of the sterilized milk. This was incubated at 37 C. for fifteen to eighteen hours when it was sent to the

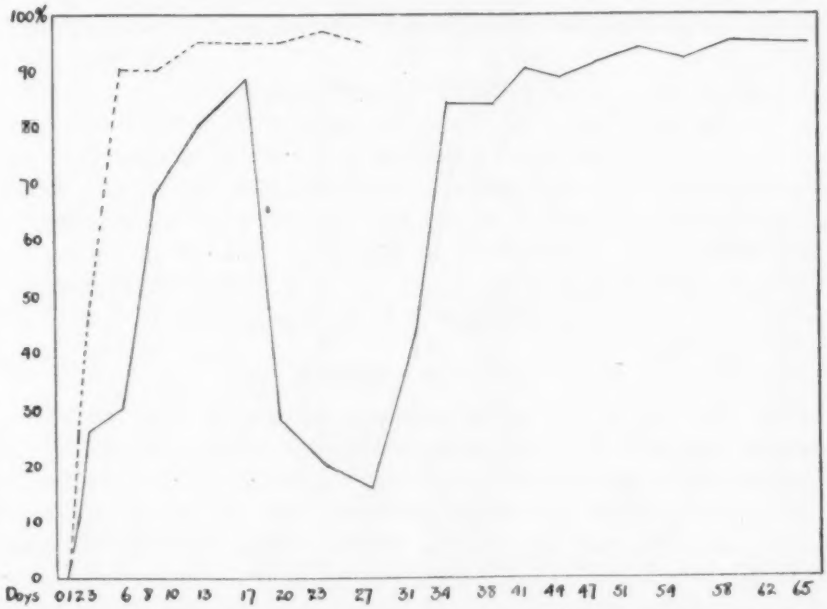


Chart 1.—Composite curve showing the percentage of *Bacillus acidophilus* in the stools during the study. Continuous line, patients with psychoses; broken line, a normal control.

wards for administration. The milk was fed for a period of sixty-four days. One liter a day was distributed for each patient, and was administered in three portions—one after each meal. No change in diet was made as the experiments of Rettger and Cheplin indicate that diet does not affect implantation. This treatment was never supplemented with lactose feeding, since implantation was secured without it. The methods employed were essentially the same as those advised by Rettger and Cheplin, except where it was necessary to make minor

9. Cultures of B. acidophilus were obtained through the courtesy of Dr. Leo. F. Rettger.

adaptations to the cases studied. Examination of the stools was continued for two weeks after the cessation of acidophilus administration, to determine how long the implantation persisted.

RESULTS

The analyses of the feces during the week prior to the administration of the acidophilus milk showed an absence of acidophilic bacteria in all patients. The stools were in general hard and well formed, and frequently showed the Welch bacillus. With the feeding of the milk, the stools remained negative for *B. acidophilus* until the second day, when this organism was recovered. The results have been plotted and are shown in composite form in Chart 1.

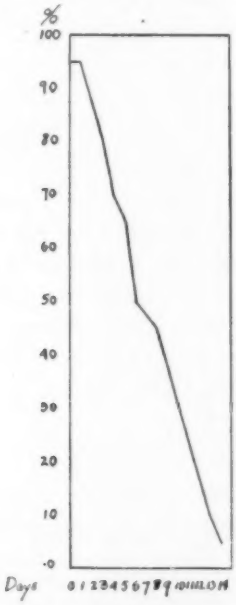


Chart 2.—Reduction in the numbers of *Bacillus acidophilus* after cessation of the feeding experiment.

It will be seen that the acidophilus organism made its appearance on the second day, and gradually increased in numbers until the seventeenth day when an average of 88 per cent. was reached. At that time, the curve took a sudden drop and decreased for about ten days to a minimum of 17 per cent. The cause of this change of intestinal flora is problematic. We attributed the diminution to one of two reasons: either the milk was no longer fed, or the stools were kept too long before being delivered to the laboratory. We are inclined to believe that the milk was omitted from the patient's diet. Difficulty was encountered in obtaining the cooperation of our patients, and it is

possible that they disposed of the milk in some way other than by ingestion. The decrease in numbers of *B. acidophilus* continued for eleven days, when the curve began to rise and in a few days reached a maximum of from 90 to 95 per cent. where it remained for the duration of the investigation.

Bacillus welchii was no longer demonstrable in the specimens which contained *B. acidophilus* and the stools lost their putrefactive appearance. Milk cultures made from suspensions of feces were indistinguishable from twenty-four hour milk cultures of pure *B. acidophilus*, which indicates the completeness of acidophilus implantation.

We were unable to correlate any change in the mental condition of the patients with the feeding of acidophilus milk during the period of the experiment.

Chart 2 shows the persistence of *B. acidophilus* in our series. It appears that after two weeks' time, the recoverable number of *B. acidophilus* reaches a minimum of from 5 to 10 per cent. of the cultivatable bacteria; the administration of acidophilus milk must therefore be continued to maintain the implantation. The results obtained in Cases 4, 8 and 9 are of significance in another way. These three subjects had, at the beginning of our study, an acneiform eruption which improved with the implantation of *B. acidophilus*.

REPORTS OF CASES

The ten cases chosen for study all presented marked constipation and frequently complained of headache and fatigue. One case showed marked secondary anemia. Several had a wide spread acneiform skin eruption. Urinalyses were negative with the exception of positive indican reactions in Cases 1 and 9. Nothing of importance was noted in the blood counts except the usual tendency toward a mild lymphocytosis. The basal metabolism rate in Case 5 was reported as + 24. Spinal fluid findings were negative. Blood urea determinations were normal. All Wassermann reactions were negative.

No changes were made in the routine ward diet during the feeding of acidophilus milk. The patients, with a few exceptions, did not complain of this procedure although toward the end of the period of feeding, several expressed some discontent at the large quantity of milk that had been offered them. Other ward patients with similar mental states were used as controls.

CASE 1.—E. S., a girl, aged 15, was admitted Sept. 29, 1921, and was discharged in improved condition May 25, 1922; the diagnosis made was: dementia praecox, catatonic type.

The patient was excited, combative, and refused to eat, asserting that her food was poisoned. Her conversation at times was incoherent and she exhibited verbigeration. There were stereotyped movements and periods of causeless laughter; auditory hallucinations also were present.

Course.—Periods of excitement alternated with periods of stupor, in which catalepsy, with its characteristic spring resistance and rigidity, were observed. Gradual improvement began after three months' stay in the hospital.

The patient was given acidophilus milk throughout the entire period of the experiment from February 5 to April 8 inclusive. This was associated with alleviation of constipation, and for three days there was a tendency to diarrhea. The patient gained twelve pounds during this period of feeding. We cannot be sure, however, that the mental improvement was associated with this procedure; she was considered a favorable case from the time of admission. Two months after discharge, when examined in the outpatient department, there had been no return of constipation, and the patient showed continued improvement. She had only partial insight into her mental condition.

Case 2.—V. S., a married woman, aged 32, was admitted Jan. 9, 1922, and was discharged improved April 30, 1922. The diagnosis was: schizophrenic reaction.

When admitted, the patient smiled and laughed in typical fashion; she was very obscene and untidy; experienced hallucinations, and at times was resistive. She showed a definite ambivalent tendency towards her husband (both love and hate) and had poor insight.

Course.—There was little improvement during the first two months in the hospital; later, the hallucinations disappeared, and periods of excitement terminated quickly after the use of continuous tubs. She began to show more interest in her surroundings, and cooperated in occupational therapy. At the time of discharge, she had only partial insight into her condition.

This patient was given acidophilus milk throughout the experimental period from February 5 to April 8 inclusive. One day she showed a tendency toward diarrhea. She lost five pounds during the period of observation. However, she had a tendency to obesity, and was in much better condition physically at the time of discharge; the constipation was definitely relieved.

Case 3.—A. B., a married woman, age 27, was admitted Jan. 14, 1922, and the diagnosis was: schizophrenic reaction. She improved and was discharged April 4, 1922.

The onset followed desertion by her husband when she became indifferent, apathetic, and at times impulsive. She complained of feeling doped and that people read her mind, though there were no hullucinations. She was in fair physical condition.

Course.—The course was characterized by gradual improvement under treatment up to the time of discharge, when the patient had good insight into her condition. Acidophilus feeding in this case likewise resulted in the elimination of constipation, and an improvement of the general physical condition. The patient gained five pounds during the period of feeding, from February 5 to 16 when she was transferred from the department.

Case 4.—M. C., a single woman, aged 25, was admitted Jan. 17, 1922, and discharged improved Feb. 28, 1922. The diagnosis was: schizophrenic reaction. The onset of the psychosis was in June, 1921, following the loss of her position. The patient developed ideas of reference, became silly and was antagonistic toward a sister. She frequently refused to eat, because the "food was poisoned." At times, she was untidy and childlike. She experienced hallucinations and had ideas of death and vague sexual phantasies. She presented a typical seclusive personality make-up.

Course.—Gradual improvement occurred during the last three months in the hospital. She was taught facts concerning sexual matters, and became less jealous of her sister; she had excellent insight at the time of her discharge when arrangements were made for return to her native country.

The patient was fed acidophilus milk throughout her stay in the hospital with resultant elimination of constipation. An acne-like eruption disappeared. The treatment in this case was considered to be very helpful, and an excellent adjunct in the treatment which was instituted from the psychogenetic viewpoint. The acidophilus milk was administered from February 5 to April 8, inclusive.

CASE 5.—A. W., a married woman, aged 47, was admitted Jan. 15, 1922, and discharged May 20, 1922, improved. The diagnosis made was: manic-depressive insanity (atypical manic phase).

On admission the patient showed marked pressure of speech and activity interspersed with periods of anxiety and depression. She had been running around the street in her night clothes; was very noisy and at times, destructive. She was frequently observed in the act of burning old clothes in her yard, and neighbors were apprehensive lest she set fire to her property.

Physical Examination.—Revealed a thyroid toxicosis; lobectomy had been performed before admission. The Goetsch epinephrin test was negative; the pulse averaged from 90 to 100; the basal metabolism rate was +24; all eye signs were positive; typical fine tremors of the hands were present; but there was no loss of weight. The patient was sent to a surgical ward, but the consulting surgeon stated that operation was not indicated.

Course.—During her stay in the hospital she showed little improvement. She presented a picture of an atypical affective disorder, with predominance of a manic reaction. Acidophilus feeding resulted in a gain of three pounds, but no other change was noted. Constipation had never been a troublesome factor. Acidophilus milk was administered from February 5 to April 8, inclusive.

CASE 6.—A man, aged 23, laborer by occupation, was admitted Feb. 1, 1922. He was adjudged insane April 29, 1922, as he had not improved. The diagnosis was: dementia praecox of catatonic type.

This patient showed a typical catatonic reaction, characterized by negativism, complete mutism and catalepsy. He frequently required tube feeding. His physical condition was good with the exception of general undernutrition and marked constipation.

Course.—The course was characterized by no improvement. Feeding of acidophilus milk resulted in clearing up the skin condition and prevented further emaciation; in spite of irregular and enforced nourishment, he did not lose weight. Acidophilus milk was administered from February 10 to March 28, inclusive.

CASE 7.—F. W., a man aged 34, was admitted Jan. 8, 1922, and discharged April 27, 1922, without improvement. The diagnosis was: dementia praecox.

A history was given of two previous confinements with a similar mental disorder. The patient maintained that he was Jesus Christ; a creator, and had unusual powers to control the destiny of the universe. He spoke of occult phenomena and gave frequent dissertations on the cosmos and theosophical matters. He had some paranoid ideas, centered about a certain individual whom he blamed for his father's financial ruin.

Course.—No change was observed in the patient's mental condition during his stay in the hospital. He never experienced hallucinations and showed no

change in the delusions mentioned. He had no insight into his mental condition. Physically he was well except for marked constipation. He obtained relief from this latter symptom, and gained in weight, after the administration of acidophilus milk, which was given from February 5 to April 8, inclusive.

CASE 8.—D. T., a man, aged 25, was admitted Jan. 19, 1922, and was committed March 15, 1922, as he had not improved. The diagnosis was: dementia praecox.

The onset of the psychosis was in September, 1920, when the patient began talking and smiling to himself and became impulsive and quarrelsome with his associates. He later developed gonorrhea and while under treatment became very excited and experienced hallucinations. The excitement alternated with periods of mutism and catatonia; at these times he refused to eat, and required tube feeding.

Course.—The patient continued to be apathetic and resistive, with periods of stupor and required tube feeding. At the time of commitment, no mental improvement was noted. Acidophilus milk was given irregularly to this patient. However, a marked change appeared in the acneiform eruption on his face and back, which cleared up considerably. His weight remained stationary. Other patients in the ward in similar condition showed a tendency to lose weight. Acidophilus milk was administered from February 5 to March 21, inclusive.

CASE 9.—R. S., a woman aged 36, was admitted Jan. 10, 1920, and discharged Feb. 18, 1920. The diagnosis made was: delirious reaction.

On admission the patient was very apprehensive and was experiencing visual and auditory hallucinations. She was agitated, restless, resistive and disoriented. During the first month in the hospital, she developed paranoid ideas about the physician who performed the lumbar puncture. Physically, she showed: marked secondary anemia; positive indican reaction; a trace of albumen in the urine, and marked constipation. A marked acneiform eruption was present over the face and back. Acidophilus milk feeding resulted in the acneiform eruption clearing up entirely. She gained ten pounds in weight and constipation was alleviated. The patient had diarrhea on one day during the period of treatment. At the time of discharge, she had from one to two normal bowel movements daily. Acidophilus milk was administered from February 5 to February 21, inclusive.

CASE 10.—G. B., a man aged 37, foreman by occupation, was admitted Jan. 30, 1922, and was committed March 13, 1922, as he had not improved. The diagnosis made was: depressive reaction. The patient was tearful, agitated and expressed many hypochondriacal complaints, with marked self-condemnation. He had attempted suicide by hanging before admission. The onset of the trouble was a reaction to financial reverses, domestic infelicity, and change of position. Acidophilus milk was administered from February 21 to March 10, inclusive.

COMMENTS

The chief aims of our experiments were: first, the relief of constipation by implantation and maintenance of *Bacillus acidophilus* in the intestine; and second, observance of the effects on the course of the psychosis. Constipation, with or without putrefaction, is frequent in patients with psychoses. When the sedentary life and frequent food refusal of these patients is considered, constipation may obviously be a result or a concomitant of the psychosis. The constipation, with what-

ever putrefaction there may be, can be corrected by *B. acidophilus* implantation and this condition may be maintained. In our series of cases this change of intestinal flora was not associated with a change in the mental condition.

We think that Holmes and Rettinger have not demonstrated an etiologic relationship between *B. aminophilus* fermentation and dementia praecox. We made no tests for the presence of *B. aminophilus,* but after the establishment of *B. acidophilus,* the few colonies of other organisms that appeared in cultures were not of the aminophilus type. We do not believe that colectomy, advocated by Cotton, is justifiable. Putrefaction, if present, may be eliminated by a change in the intestinal flora. Although the administration of acidophilus milk did not improve the mental condition of our patients, we believe that it may be employed with benefit during a psychosis because of the resultant relief of constipation, the improved appetite, and the gain in weight.

CONCLUSIONS

1. Implantation of *Bacillus acidophilus* in the intestine of patients with psychoses by the oral administration of acidophilus milk resulted in elimination of intestinal putrefaction and in some cases in an increase in weight but no improvement in the mental condition.

2. Intestinal putrefaction is probably a concomitant of some forms of psychosis if it is at all related.

3. The administration of acidophilus milk is recommended in the treatment of psychoses as a means to physical betterment.

Obituary

EDWARD MERCUR WILLIAMS, M.D.
1881-1923

In the death of Dr. Edward Mercur Williams, the American Neurological Association has lost one of the most promising of its younger members, one who bade fair to become a leader in American neurology. It is fitting that I should place on record an estimate of his acquirements and character as, for three years, from 1909 to 1912, he was my private assistant and associate, and was also a valued member of the neurologic staff of the medical school of the University of Pennsylvania. Thoroughly trained in general medicine and in neurology, honest in his convictions and methods, he was courteous, kindly and unobtrusive. He was a gentle soul, although one who, when necessary, could stand firmly for his own position and opinions. He became a member of this association in 1912 and contributed several valuable papers to our proceedings.

The parents of Dr. Williams were Horace Guernsey Williams and Ann Bowkley Williams. He was born October 16, 1881, at Wilkes Barre, Pa. From Wilkes Barre, the family moved to Lykens, Pa., situated in a region noted for a highly prized variety of coal. For several years, his father, a distinguished mining engineer, was interested in operations in the Lykens Valley for the Pennsylvania Railroad. While at Lykens, Dr. Williams went to the neighborhood school.

In 1895, he went with his family to St. Davids, Pennsylvania, and became a student at the Radnor High School, where he graduated in 1900. After taking a special course in biology in the University of Pennsylvania, he entered the medical school, graduating with a good record in 1905. Soon after graduation he became an intern in the Monongahela Surgical Hospital, in the western part of Pennsylvania, where he remained until some time in 1906. After leaving Monongahela, he came to Philadelphia and took a postgraduate course in the Polyclinic Hospital School.

In 1907, he went abroad to complete his medical studies and selected Vienna as the first stopping place in his foreign postgraduate pilgrimage. Here he remained for almost two years, working in the laboratories of Professor Obersteiner, Frankel Hochwart, Marburg and Ludwig Braun. While in Vienna, he became highly expert in the cutting, staining and studying of specimens illustrating diseases of the

EDWARD MERCUR WILLIAMS, M.D.

nervous system. He completed his foreign studies in Paris, chiefly under the direction of Pierre Marie. In Paris, he also made general use of the clinics, hospitals and societies.

While abroad, Dr. Williams published several papers, two in German,—"Vergleichend anatomische Studien über den Bau und die Bedeutung der Oliva inferior der Säugetiere und Vögel" (Concerning the Anatomic Structure of the Inferior Olive in Mammals and Pigeons); and "Ueber Fazialislähmung nach Zahnextraktion" (Facial Paralysis Following Extraction of the Teeth).

The first of these papers was written under the stimulus and supervision of Professor Obersteiner, and the second was from the Vienna Medical Clinic of Professor v. Noorden. Both of these contributions contained original observations of sufficient value to merit the approval of the distinguished group of neuropathologists of Vienna in whose laboratories Dr. Williams spent most of his working hours.

Dr. Williams returned to this country in August, 1909, when I first made his personal acquaintance. He soon became a member of the neurologic staff of the medical school of the University of Pennsylvania, in which he held several important positions, among these being, Instructor in Neuropathology, Assistant Neurologist and Assistant Physician in the neurologic dispensary to the hospital of the University of Pennsylvania. In all of these positions he did excellent work. During his connection with the University Medical School, he published several important papers—"Chronic Hypertrophic Spinal Pachymeningitis" (with Dr. Charles K. Mills), "État Vermoulu," "Intermittent Claudication in the Upper Extremities," "Cerebral Rheumatism" and "Typhoidal Hemiplegia."

Dr. Williams moved to Vinita, Okla., in November, 1912, becoming the resident physician of the East Oklahoma Hospital for the Insane. Here he did excellent work for about one year, after which he removed to Sioux City, Iowa. In Sioux City, he engaged in private work, chiefly as a neurologic consultant, devoting himself assiduously to practice, research and clinical investigation. In addition to his work as a neurologic consultant, he became well known in the courts as a medical witness in cases involving insanity and traumatic and other affections of the brain and nervous system. Dr. Williams soon became well known throughout the middle west, his medical work extending to various localities in Iowa, Nebraska, Minnesota and South Dakota.

In the preparation of this sketch, many letters of condolence and of appreciation from members of the medical and legal professions have come under my notice. These are of the same general tenor. The letters from his medical confreres lay stress on his thoroughness, efficiency, honesty and reliability. They speak of the constant use

made by him of the technical knowledge obtained by him in the laboratory. The tributes from the legal profession all call attention to his modesty, sincerity and learning, and to the clearness, precision and truthfulness of his testimony. His standing in the courts as testified to both by members of the bench and the bar was of the highest character.

Dr. Williams' career in Sioux City extended over a little more than nine years.

His health was never very good. He had some form of cardiac disease, and while he was fond of outdoor life, he was compelled to restrict his exercise.

In January, 1923, he had an attack of influenza, which was followed by pneumonia. He went to St. Joseph's Mercy Hospital in Sioux City, and although he received every attention from his medical brethren and others connected with the hospital, he died January 5, 1923. His death not only left a void in his family and among his friends, but was the subject of marked regret among the profession of Sioux City and the state of Iowa. Resolutions of appreciation and regret were adopted by the Iowa State Medical Society.

During the short period of about fourteen years between the time of his return from Europe and his death, besides the contributions already mentioned Dr. Williams published a considerable list of papers, some of which were of unusual value, both from a clinical and pathologic standpoint.

In 1911, he married Florence Henry Lofland, of St. Davids, Pa. He is survived by his devoted wife and one child, Sarah Lofland Williams, born in 1916.

When one in the fulness of years departs from our midst, we may have regrets that we can no longer be cheered by his presence, and in that presence can no longer be stimulated by the story of his well wrought career; but when the passer is one stricken when he had scarcely reached his prime, one who gave promise of much to come, then are our hearts filled with a peculiar sadness, begotten by the thought that he was cut off too soon for him to achieve his soul's desires, and yet, we are gladdened when we feel that such opportunities as he had were not neglected and that the results he obtained are worthy of our admiration.

CHAS. K. MILLS.

News and Comment

THE THERAPY OF EXOPHTHALMIC GOITER

Exophthalmic goiter has long offered a problem of serious import to the clinician, since it causes much immediate distress to the patient and presents uncertainties of prognosis in most cases. Until recently, errors in diagnosis were frequent, for few clearly defined aids to clincial judgment were available. Now that there is more agreement in respect to the distinguishing characteristics of exophthalmic goiter, the therapy of the disease has become the subject of active discussion. In a recent issue of the *Archives of Internal Medicine*[1] is presented the outcome of serious researches by competent investigators which emphasize the conflicting points of view.

The crux of the situation involves the natural or spontaneous course of the disease. This is admittedly fundamental to any final evaluation of specific measures of treatment that may be undertaken. It is often alleged that the natural course of exophthalmic goiter is a most uneven one, and that improvements and relapses may occur at any time spontaneously. Means and Holmes of Boston have wisely remarked, however, that until some one produces a satisfactory experimental exophthalmic goiter, controls will be secured only by chance. Under such circumstances, therefore, it will not be easy to withhold promising treatment from patients, even if a considerable element of uncertainty as to its fundamental value may exist.

Recent treatment of exophthalmic goiter has for the most part proceeded on the theory that it really represents an aspect of hyperthyroidism. Means and Holmes remark that if we accept the theory that the symptoms manifested in hyperthyroidism are due to increased activity of the cells composing the thyroid gland, then the application of any method of treatment which is known to inhibit or destroy cell function would be rational. Surgery and roentgen-ray treatment have accordingly vied with each other for favor in bringing about the desired result. Rarely has it been possible to study such an array of carefully collected data as the intelligent collaboration of the internist, the surgeon and the roentgenologist has made possible in the study of a large number of patients at the Massachusetts General Hospital by Means and his collaborators. About two thirds of these cases exhibiting unmistakable exophthalmic goiter showed either recovery or improvement coincident with treatment by suitable irradiation with roentgen rays. The Boston clinicians assert that in exophthalmic goiter, when treated by the roentgen ray, if good results are not secured in a few months, surgery should be employed. Prolonged roentgen-ray treatment in patients showing no response is undesirable. They also believe that some patients with exophthalmic goiter who are not

1. Means, J. H., and Holmes, G. W.: Further Observations on the Roentgen-ray Treatment of Toxic Goiter, Arch. Int. Med. **31**:303 (March) 1923. Kessel, Leo; Lieb, C. C., and Hyman, H. T.: Studies of Exophthalmic Goiter and the Involuntary Nervous System: Kessel, Leo; Hyman, H. T., and Lande, Herman: III, A Study of Fifty Consecutive Cases of Exophthalmic Goiter, ibid. **31**:433 (March) 1923.

cured by the roentgen ray are, perhaps, made better operative risks by it. A combination of the two forms of treatment may sometimes accomplish more than either does alone.

In contrast with this is the report of the course of fifty cases of fully developed exophthalmic goiter observed at the Mount Sinai Hospital, New York, by Kessel, Hyman and Lande. The course of the illness was followed with as little interference with the natural tendency of the disease as was compatible with the comfort of the patient. At any rate, "specific" therapeutic procedures were not instituted. Such observations on patients kept essentially at rest alone may quite properly be regarded as a control investigation for comparison with the result attributed to irradiation and surgery. In the opinion of the New York clinicians, the spontaneous course of exophthalmic goiter is toward arrest in the vast majority of cases. In patients who develop the disease late in life (after 45 or 50) the prognosis is poor. If these cases are excepted, the prognosis is excellent under a regimen of "skilful neglect." To establish the efficacy of any specific therapeutic measure, we are told further, one should demand that definite proof be offered that the results obtained are better than those reported here of the "spontaneous" course of the disease.

All investigators now agree that until a specific diagnostic test is discovered, no reported case of exophthalmic goiter should be accepted as a genuine case unless the basal metabolism is distinctly and repeatedly elevated. According to the New York clinicians, by this alone can the disease be differentiated from "autonomic imbalance." In the stage of arrest, differentiation can be made only by the history of the crisis. The clinical picture varies as the age of the patient at the time of onset of the disease. In other words, the variation, especially in regard to the presence and extent of the exophthalmos, is dependent, we are told, on the end tissues of the patient, rather than on the specific elaboration of any toxic product. It may be well to reiterate here that thyroid hyperplasia and thyroid adenoma may exist for years without at any time causing sympathomimetic symptoms or alteration in metabolism. Kessel, Hyman and Lande insist that in exophthalmic goiter the dominant derangement is in the realm of the involuntary nervous system. This may not be primary; but the primary cause, whatever it is, must at least operate through the mediation of the involuntary nervous system. Means and Holmes point out, further, that, in toxic adenoma, roentgen-ray treatment appears to effect a similar improvement to that noted by them in exophthalmic goiter; but so far they have used irradiation only with patients who have refused operation. In toxic adenoma, in contrast to exophthalmic goiter, surgery probably removes the actual cause of the disease, the adenoma. The indication for surgery, they add, would, therefore, seem more definite than in exophthalmic goiter. Even in toxic adenoma, however, in certain cases that are too thyrotoxic for safe operation, the roentgen ray may be used to advantage. In the light of present-day evidence, the choice of therapeutic procedure presents, indeed, a difficult perplexity.—*Jour. A. M. A.*, April 14, 1923.

Abstracts from Current Literature

RESEARCHES ON THE ABDUCENS NERVE AND PARTICULARLY ON THE SIGNIFICANCE OF ITS ACCESSORY NUCLEUS. T. TERNI, Folia Neuro-Biolog. **12**:277, 1922.

The author refers to the first researches of Lugaro and Pacetti after whom the "nucleus accessorius abducentis" was named, and to the work of later investigators: Siemerling and Boedeker, Giannuli, Panegrossi, Van Gehuchten, Kaplan and Finkelburg, Bach, Tsuchida, Von Falkenburg, Jacobsohn, Kappers, Cajal, Fuse, and Sterzi, whose conclusions on the existence of an accessory nucleus of the sixth nerve in vertebrates are contradictory.

In order to clear up the morphology and possibly the functional significance that the accessory nucleus possesses in the constitution of the sixth nerve, the author has undertaken the investigation reported in this paper. He has endeavored to find first, the central origin of the abducens in the representatives of those groups of animals (fishes, reptiles) in which no duplicity of the nucleus has been demonstrated; and secondly, to define with more detail than has been done by others, the constitution and the central relations of the sixth nerve in the representatives of animals (birds, mammals), in whom scanty observations had led to positive preliminary results. His material for investigations were mainly embryos in which the application of Cajal's method for neurofibrils gave excellent results.

For the researches on the trout, the swimming embryos of *Salmo fario* and *Salmo carpio* from 10 to 20 mm. long, were used; serial sections were cut transversely.

For the investigation in *Gongylus ocellatus,* there were utilized ninety-seven embryos in different stages of development from the very early periods up to birth, cut in series in different planes. The greater number of embryos corresponded approximately to the same stages already described by the author in one of his works on the spinal cord. Besides, a few series of embryos fixed in Zenker's fluid and stained with alum-carmine were made. Serial sections of brains of new-born *Gongylus* stained with Cajal's method, and of adult *Gongylus* stained either with Cajal's, or with Weigert's or Nissl's methods were also prepared.

For the investigations on chickens, several embryos at different stages of development, impregnated with Cajal's method, were used. One series of chicks was prepared with the Cajal method and one with the Weigert method. Adult animals were also dissected for the purpose of becoming oriented on the muscles supplied by the sixth nerve.

The observations on *Mus rattus* were conducted on embryos, whose brains were cut transversely and longitudinally and stained with Cajal's method.

The results of the author's investigations on the central origin and on the peripheral distribution of the sixth nerve are preceded by the most important bibliographic data on the subject. Also, some personal contributions to the development of the nerve, and some information concerning the morphology of the third eyelid (an organ which is directly or indirectly operated by muscles supplied by the abducens), are given.

The findings of the author are:

1. *Fishes.*—Trout: Cajal's and Tello's findings on the positions of the nucleus of origin of the abducens are confirmed. An accessory nucleus of the abducens, situated laterad and distant from the principal nucleus does not exist. A third eyelid and a muscle retractor of the ocular bulb, differentiated as a special muscle, is also lacking. The abducens in its peripheral distribution is clearly seen to reach a single muscle, the external rectus.

2. *Reptiles.*—Gongylus: In embryos at the stage 4 (corresponding to stage 123 of Peter in *Lacerta a.*) the principal nucleus of the abducens is situated, as in all tetrapoda, very dorsad in the bulb, almost in contact with the ventral and lateral surface of the fasciculus longitudinalis medialis. It forms a small continuous column, which goes from a level corresponding to the caudal pole of the motor nucleus of the fifth nerve, to a point very near to the rostral pole of the motor nucleus of the facial. The cells are fairly large, appear like stars, having dendrites more developed outwardly, and which stain poorly with silver. The neurites describe a slight arch thus causing the root of the abducens to emerge somewhat far from the middle line. Not all the radicular fibers originate from the cells of the principal nucleus. In the root of the sixth nerve one may clearly see fibers that come from another region of the rhombencephalon. These fibers are very large; they run in bundles or singly and form an elbow in correspondence with the principal nucleus, and then follow the same direction as the nerve in their common exit from the rhombencephalon. These fibers originate from a group of cells very characteristic in shape, which are situated in a ventrolateral position, mediad to the descending root of the trigeminal. This nucleus, never described in reptiles, corresponds to the one described by Van Gehuchten in chickens and by Lugaro in rabbits. It is a small flat column, which, from a caudal level corresponding to the rostral pole of the motor nucleus of the facial, goes a short distance below the caudal pole of the principal motor nucleus of the trigeminal. This nuclear column has approximately the same length as the principal nucleus; it is displaced slightly caudad. The cells, owing to their very typical form, are easily distinguished from the cells of the principal nucleus and from those of the other nearby nuclei. They are multipolar, very large, larger than those of the principal nucleus, and are very easily stained with Cajal's method. They present two principal groups of fine long dendrites and an axon which usually originates from the dorsal and median portion of the cell in the proximity of the nucleus, which is very large and provided with a conspicuous nucleolus.

The cells of the accessory nucleus form a very thick and well circumscribed group in the embryo. They cannot be mistaken for other near or distant elements, such as the cells of the sensory nucleus of the fifth, the cells of the nucleus of Deiter and the elements of the nucleus magnocellularis of Cajal—the latter possessing an entirely different aspect, size and distribution. The axis-cylinders run in bundles of two to four and, before entering the common root of the sixth nerve, describe an elbow around the principal nucleus. They cross the fibers of the direct vestibulospinal tract and enter, without association, among the fibers of the principal nucleus; they maintain their independence from other systems of fibers, such as the trigeminal system and the central acoustic pathway. A conspicuous group of dendrites of the cells of the accessory nucleus of the abducens, however, constantly converges toward the descending root of the trigeminal, and penetrates among its fibers. It is

likely that the accessory nucleus and the principal nucleus of the sixth nerve originate from a cell group which originally constituted a single unit.

In very advanced stages, up to birth, the transformations in the general constitution of the nucleus of the sixth nerve are very slight. At birth, the relationship between accessory and principal nucleus remains the same as in embryos, except for the neurofibrillar reaction which is less distinct. In the adult this relationship is not so easy to trace, since the Cajal method does not stain this system very electively. With the Weigert method the accessory radix of the sixth nerve is seen entering the common root, but its fibers cannot be followed to their origin from the accessory nucleus. With alum-carmine and the Nissl method the small group of cells of the accessory nucleus is found in the same position as in the embryo, namely in proximity to the descending radix of the fifth and slightly caudad to the principal nucleus of the sixth. However, the cell elements cannot be well differentiated from other systems nearby.

Here the author gives the peripheral distribution of the abducens, studied in correlation with its central configuration. He found that in *Gongylus,* the muscles supplied by the abducens correspond almost entirely to those described in *Lacerta* by M. Weber. For this study he used the Cajal method in embryos of very advanced stages—from stage 4 on. After a clear and complete description of the muscles and their nerve supply, he gives the number of fibers found in the two nerve trunks which result from the bifurcation of the abducens as 150. One hundred of these are for the external rectus, and thirty to forty fibers compose the nerve for the motor and retractor muscles of the nictitating membrane. There seems to exist a correlation between the number of the fibers of the two branches of the abducens and the number of the cells of the two nuclei of origin. The author is of the opinion that the fibers originating from the accessory nucleus are distributed to the muscles of the third eyelid (retractor and bursal), and that the other fibers from the principal nucleus, are for the external rectus. A table giving the number of cells of the nuclei and the corresponding fibers in the main trunk, and in both branches as distributed to the different muscles, in the different stages, is given. The number of cells of the accessory nucleus do not change at the different embryonal stages.

3. *Birds.—Gallus domesticus.* In embryos of chicks of 6 or more days, the accessory nucleus of the abducens is made up from a thick group of cells smaller than the principal nucleus, situated in the medulla oblongata at about the same level as the principal nucleus. It extends from the caudal pole of the motor nucleus of the facial to about the rostral pole of the dorsal nucleus of the vagus and glossopharyngeal. It is located between the medial margin of the descending root of the trigeminal and a cellular group of still scarcely differentiated elements, which the author believes to be the superior olive. The cells of the accessory nucleus are multipolar, much elongated, and possess dendrites which converge in a conspicuous group toward the lateral surface of the bulb, where the fibers of the descending root of the trigeminus course. The cells are somewhat smaller than the cells of the nucleus magnocellularis reticularis. The neurites of many reticular cells, situated in the proximity of the accessory nucleus of the abducens, approach, or cross, the direction of the accessory root of the abducens. For this reason some authors have mistaken the nuclei of the reticular substance, viz: the superior, the middle, the inferior and the disseminated, for the accessory nucleus. In more advanced stages up to the thirteenth day, the accessory nucleus becomes more con-

spicuous, but its relationship does not vary. In a series of adult chickens prepared with the Weigert method, the author has identified the nucleus in a small column of cells at the level of the principal nucleus of the sixth, near the external margin of the superior olive, and almost in contact with the medial edge of the spinal root of the fifth. In general, the accessory nucleus of the abducens and the fibers which originate from it, show an appearance similar to the formation indicated with (*a*) in the Figure 1 of Cajal (1908). Regarding the distribution of the abducens, the author confirms what has been found in the adult. In embryos of 6 days, one can recognize a distinct muscular rudiment for the external rectus and for the muscles of the nictitating membrane, and the branches of the sixth pair which supply the separate rudiments.

4. *Mammals.—Mus Rattus.* Extensive investigations on the accessory nucleus of the sixth nerve could not be carried out, because in the embryos of these animals the Cajal reaction does not give as good results as in reptiles and birds. The accessory nucleus of the abducens is made up of a thick celi group, well separated from the motor nucleus of the facial and from the principal nucleus of the abducens, situated at about the same level as the latter but much more ventrad and laterad. Its position is between the nucleus of the facial and the principal nucleus of the abducens, but much nearer to the former, and well distant from the descending root of the fifth. The cells are stellate, long, but smaller than the cells of the substantia reticularis grisea.

The results of some simple physiologic experiments on the trigeminal-abducens reflex, obtained by the author, harmonize with the anatomic findings. In reptiles, a very brisk reflex of the third eyelid is found. It consists in the immediate closure of the lid on mechanical or electrical stimulation of the peripheral territory of the trigeminal. At the same time a slight enophthalmos is observed without rotatory movement of the ocular bulb. After decerebration and destruction of the optic lobes, the reflex persists unchanged. In chickens and in other domestic birds the reflex has also been found by the author, but it is not accompanied by exophthalmos. (These experiments were reported in Archivio de Fisiologia, **20**:4, 1922.)

In discussing the morphologic and functional significance of the sixth nerve and its accessory nucleus, the author concludes that although this nerve, in a large number of vertebrates, supplies more than one single muscle, and, although it originates from a nuclear complex whose accessory constituent is situated quite laterad and ventrad in the medulla oblongata, yet it presents the singular appearance of a ventral spinal root. The accessory nucleus of the sixth nerve supplies those muscles which either directly or indirectly are connected with the movements of the third eyelid. The grouping of the radicular cells of the abducens in two nuclei, distinct and very far from each other, is in perfect accord with the doctrine of Kappers, according to whom a motor nucleus tends to approach the system from which it receives (or will receive later) the greatest amount of functional stimulus. It is likely that the accessory nucleus will make important connections with the different pathways making up the descending root of the trigeminal, inasmuch as the movement of the nictitating membrane is closely related to stimuli from the territory of the peripheral distribution of the trigeminus. The principal nucleus remains, for obvious reasons, near the systems of the optic and acoustic pathways, a portion of which run in the medial longitudinal fasciculus.

NACCARATI, New York.

SPIROCHETES IN SYPHILITIC ENDARTERITIS OF THE BRAIN.

F. Sioli, Arch. f. Psychiat. u. Nervenkrankh. **66**:318 (Oct. 18) 1922.

In this article Sioli discusses the general problem of so-called metasyphilis, his general thesis being that it is premature to make any very definite theoretical assumptions until more work has been done to show the relation or non-relation of the spirochetes to the pathologic processes. He first calls attention to the fact that the idea of general paralysis as a syphilitic disease depending on the effect of syphilitic toxin without the spirochetes in the brain, was upset by the work of Noguchi and Moore and their followers. In cases of endarteritis of the small cortical vessels, spirochetes have not been found in the past, which led to the hypothesis that the endarteritis was the result, not of the local spirochetal activity, but of a toxic process. This has been the general idea of the metasyphilitic processes and therefore it becomes of great importance to determine the presence or absence of spirochetes in cases of endarteritis which will again illuminate the whole metasyphilitic problem.

The endarteritis of the small brain vessels was first described by Schüle in 1872 and its histologic characteristics were established in 1903 by Nissl on the basis of four cases, three of which had been observed by Alzheimer and discussed under the title "Diffuse Brain Syphilis Without Adventitial Sheath Infiltration" or again as "A Non-Gummatous Form of Brain Lues." Nissl characterizes the process as follows: The change is diffuse; adventitial sheath infiltration does not occur except for an occasional mast-cell. The cells of the intima are greatly proliferated and there is considerable increase of the cells of the intima which may divide the vessel lumen into several channels. As a result of the proliferation of the endothelial and adventitial cells, the muscle coats are disorganized. New vessels are formed by outgrowths of the old, and the elastic coat is broken up. There is a relatively small amount of disorder of the tangential fibers and supraradial network. Glia proliferation occurs, but of a type different from that found in general paralysis, namely, a growth of the protoplasm with very little fiber formation and with increased size of the glia nucleus. The nerve cells in the chronic form of this disorder show an enormous swelling of the whole body with a tendency to disintegration of the cell. In all of the cases one finds the Heubner type of endarteritis in the vessels of the base of the brain without adventitial infiltration and without mesarteritic changes.

In 1904, Alzheimer described three more cases and gave as one of the essentials, disorganization and intertwining of the coats of the vessel wall resulting from a marked proliferation of the cells of the vessel wall. All three of these cases showed microscopically, small areas of softening.

In 1920, Jakob made a study of twenty cases of endarteritis, of which thirteen were pure cases without infiltration or inflammatory changes, while in seven there was a combination of endarteritis of the small cortical vessels with meningitis or diffuse infiltrative changes similar to those of general paralysis. Jakob found that in almost all cases there was some softening or areas of degeneration, which he believes are the result of arteriosclerotic changes and not essentially specific, agreeing with the concept previously enunciated by Schröder. Jakob agrees essentially with Nissl and Alzheimer that there is a type of syphilitic endarteritis of the small vessels of the cortex characterized by: the absence of lymph sheath infiltration; a marked increase in cells of the vessel wall, with a wiping out of the separation between the

coats of the vessel wall; a considerable degree of proliferation of the protoplasmic elements of the glia cells; and a relatively small amount of diffuse swelling of the ganglion cells and nerve fibers, of irregular distribution over the cortex, with localized increase of the process here and there.

The important point of Jakob's work is that he was unable to discover spirochetes in any of these cases of pure endarteritis. He therefore believes in the theory that the process is of toxic genesis. Jakob further describes one case in which the frontal portion of the brain showed small vessel endarteritis, while in the hind half of the brain a marked infiltrative process was in evidence. In the portion without infiltration he was unable to find spirochetes, whereas in the hind part of the brain where infiltration was considerable, spirochetes were found.

Hauptmann emphasizes this finding of Jakob in building up his theory concerning the pathogenesis of syphilitic and paralytic brain changes. Hauptmann believes that infiltrative appearances speak for the local presence of spirochetes, whereas parenchymatous degeneration and endarteritis suggest a toxic genesis. In other words, from the bacteriologic standpoint, Hauptmann postulates a non-specific process, one that is the result of toxicity of a protein nature, not depending on the presence of spirochetes in the central nervous system.

After reviewing the foregoing, Sioli presents a case of endarteritis in which he found spirochetes. As this has so much importance in relation to the metasyphilis problem, it is presented in some detail.

The patient was born in 1872 and died in 1919. He was picked up on the street confused, disoriented, and grandiose. The pupils were unequal and one did not react to light; the tongue protruded to the left with tremor; the knee jerks were very lively but there was no Babinski nor Oppenheim sign; there was slight speech defect; the heart was increased in size but the heart sounds were clear; the second aortic sound was increased; the Wassermann reaction was positive in the blood and spinal fluid; globulin was present and there was a marked lymphocytosis in the fluid. The man remained euphoric, demented, suggestible, and full of ideas of grandeur; he rapidly deteriorated physically and mentally. He had five mild epileptiform seizures and died in 1919.

At the necropsy there was a fresh hemorrhagic pachymeningitis as well as edema of the pia and chronic leptomeningitis of the convexity. The brain weight was 1,420 gm.

Sections from the central convolution showed increase in size of the vessels, with proliferation of the cells of the vessel walls and an increased number of large, poorly staining nuclei. In many places the layers of the vessel wall could not be distinguished. In several places there was increased vessel formation, which, however, was not general. Rod cells were found sparingly through the cortex. In the adventitial sheath no plasma cells were found. There were no true lymphocytes and only an occasional mast cell, and in several places granular cells with green pigment or basophil-metachromatic content. Likewise in the pia of this region no plasma cells or lymphocytes were found. The ganglion cells were markedly changed with all varieties and degrees of disorder.

The section from the frontal convolution showed a considerable thickening of the pia and a disturbance of the architectonics of the cortex. The endarteritic condition was more marked than in the central convolution. An extraordinarily marked change in the ganglion cells was found. There was

more glia than in the central area with increase in the size of the nuclei and protoplasm and an increased number of glia cells especially about the ganglion cells. Many typical rod cells were found. Around a number of the larger and smaller vessels of the cortex several plasma cells were observed and these were also found in the pia. Further study showed that plasma cells were occasionally present in the frontal and occipital convolutions and in the gyrus rectus.

Spirochetes were found in the basilar artery. They were also found in several places in the central convolution, in the first frontal convolution, and in the gyrus rectus. The basilar artery showed proliferation of the intimal coat, with disorder of the elastica, and in this area colonies of spirochetes were found. These spirochetes were especially fine and thin, the so-called "brown spirochetes."

Aside from these local spirochete colonies, organisms were also found along the entire wall of the basilar artery. In addition, some were found in the coagulated blood within the basilar artery. In the basal pia mater, in the region of the midbrain, several spirochetes were found, and in one small area a large mass of spirochetes was observed. They were also demonstrated in the adventitia both of the pial vessels and of the cortical vessels.

In the central convolution the spirochetes were so few that one could not speak of their relation to the tissue or vessel walls. In the first frontal convolution many spirochetes were present, not diffusely scattered, but in a great many localized areas, so that in almost the entire frontal cortical area spirochetes could be found. They were present in the entire brain tissue but not in such close relation to the vessels as Sioli has described in general paralysis. However, the majority were in the neighborhood of blood vessels, and many were found in the vessel walls.

In the gyrus rectus the spirochetes were less frequent than in the frontal convolution, but in greater number than in the central convolution. No spirochetes were found in the blood coagulum of the vessels of the convexity either in the pia or brain substance. Of the viscera only the tongue and aorta were preserved and in these organs no spirochetes were found.

The case showed histologically, proliferation of the intima of the basilar artery, endarteritis of the small pial and brain vessels with areas of degeneration, heaps of granular cells, areas in the ganglion cells with pericellular effects and marked general glial changes. The endarteritis was present in all portions of the brain studied. In a number of areas there was a pial endarteritis without infiltrating cells. In other places (frontal lobe) the endarteritis was combined with a small degree of plasma cell infiltration, the endarteritis being more marked in the areas where the infiltration occurred.

In general, Sioli states that the case showed the picture described by Nissl of endarteritis and not the type of general paralysis. In summing up the meaning of his findings, namely of spirochetes in a case of endarteritis of the smaller vessels, Sioli states that in three other cases he was unable to find spirochetes and therefore would draw conclusions with a certain amount of reserve. While he is unwilling to make a definite formulation, he still feels that his unique finding robs the toxic theory of much of its potency, as his finding shows that the endarteritis may be associated with the presence of spirochetes.

In regard to the finding of spirochetes in the blood coagulum of the basilar artery within the lumen of the artery itself, he raises the question of whether these were present during life, or were the result of postmortem wandering.

Because of their location in the basilar artery, he is of the opinion that they existed here during life. If this is so, it would lead to the assumption that late brain syphilis is not only the result of spirochetes which have made their way to the brain during the secondary period of the disease, but that they may also reach the brain during any period of the disease, arriving by way of the blood vessels. He maintains that an important question is still to be solved, namely, whether one can make a sharp differentiation between the definite forms of so-called metasyphilis, or whether they are not merely combinations of the same process in different anatomic locations. It seems to him that any hypothesis on this point can well wait until more fundamental work has been accomplished and in this regard he calls attention to the remarkably small amount of work that has been done on this subject.

The paper is illustrated by seventeen photomicrographs showing the histologic and spirochetal findings. It is the first contribution showing the presence of spirochetes in endarteritis syphilitica of the brain, and as such is of the utmost importance in opening a new line of approach to the question of the genesis of this condition. Unfortunately one is not thoroughly convinced that the case is not one of general paralysis complicating endarteritis. Although he states that the histology was not that of general paralysis, the clinical picture, the serologic findings, and the plasma cell infiltration of the frontal areas are suggestive.

SOLOMON, Boston.

CEREBRAL SYPHILIS AND PSYCHOSES. L. REDALIÉ, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:230, 1922.

The introduction of lumbar puncture has contributed vary largely to our understanding of the psychoses associated with cerebral syphilis. Koenigstein and Spiegel have recently studied the neuropathologic alterations to be found in patients who did not present clinical evidence of lesions of the central nervous system, but in whom the cerebrospinal fluid pointed to syphilitic involvement. In all of these cases anatomic investigation revealed areas of meningeal infiltration. This observation is of great interest and indicates the great difficulty present in the diagnosis of such conditions. On the other hand there are cases in which the coincidence of a psychosis with syphilis of the nervous system appears to be quite fortuitous.

Herschmann does not hesitate to make a diagnosis of psychogenic depression, hysteria or catatonia in individuals whose spinal fluid gives every evidence of neurosyphilis; regardless of the anatomic evidence of meningeal syphilis these psychoses are to be considered as nonsyphilitic. This is notably true in cases of manic-depressive insanity in which attacks of this psychosis may precede the syphilitic infection. It is impossible in many instances to answer the question as to whether the clinical picture is in any way modified by the presence of cerebral syphilis. Plaut believes that acute and chronic forms of psychoses characterized by hallucinations, ideas of persecution, and anxieties, without grosser evidence of disorientation or impairment of memory are characteristic of syphilis. Marcus divides these cases into types with acute hallucinations, with epileptiform seizures, and with catatonia. Other authors, including Kraepelin, speak of a paranoid, or a schizophrenoid type of cerebral syphilis.

Redalié presents histories of the following interesting cases:

1. A man, born in 1866, constitutionally somewhat depressed, but otherwise quite well. The exact time of his syphilitic infection could not be determined

but was probably not later than 1917. In 1918 his blood Wassermann reaction was positive. In the winter of 1920 to 1921 his vision began to fail, for which he consulted an oculist. A diagnosis of optic atrophy was made and an unfavorable prognosis given. He became depressed, self-accusatory, said that he was poisoning the entire world, that everything he touched became infected; discontinued his work, refused his nourishment, became agitated at night, but did not hallucinate and retained a good memory. Argyll Robertson pupils were present; the patellar reflexes were absent; lumbar puncture done on September 28 revealed positive Noguchi and Wassermann tests, 41 lymphocytes to the cubic millimeter; Wassermann test of the blood negative. He died Oct. 6, 1921. At necropsy a chronic, fibrous, leptomeningitis with lymphocytic infiltration and perivascular infiltration were noted. A syphilitic type of arteritis was observed in a branch of the anterior superior cerebellar artery. The parenchymatous structures were little involved. Pulmonary embolism and general infection were evidently responsible for death.

The interpretation of this case was difficult. One had to consider: (1) a psychogenic depression; (2) hallucinosis of Plaut or the paranoid type of cerebral syphilis; (3) tabetic psychosis; and (4) *confusion délirante* of Marcus. Redalié believes that the emotional trauma in this patient, debilitated by cerebral syphilis, was responsible for the onset of the psychosis. He does not venture to say what course this patient's illness might have taken had he (the patient) not suffered the psychic trauma.

2. A bank employee of good mentality, born in 1885, lost 40,000 francs in speculations in 1917. He became depressed and tacturn. In 1920 he consulted numerous physicians because of gastric crises and vomiting. Albumin was found in his urine. He became fatigued and discouraged and asked to be killed. On admission to the hospital in January, 1921, he seemed indifferent, lost appetite, was negativistic and had to be fed by tube. He was extremely emaciated, the pupils were unequal and irregular but reacted normally. Lumbar puncture showed 14 lymphocytes to the cubic millimeter, a positive Wassermann reaction, Noguchi test faintly positive; there was a strongly positive blood Wassermann reaction. In March an irregular temperature appeared and he died of pneumonia on March 26. Necropsy showed an osteomyelitis of the skull with involvement of the superior longitudinal sinus. The meninges were hyperemic in the frontal area and a few small hemorrhages were noted over the convolutions. There were no findings indicating a general paralysis and no plasma cells could be found. It seems that in this case the organic involvement of the brain was of greater importance than in the preceding one. The preservation of memory and intelligence is to be noted.

3. A woman, born in 1888, at the age of 9 years suffered from convulsions which disappeared after six months. She led the life of a prostitute for ten years, but denied syphilis; was imprisoned on several occasions; drank one to two liters of wine and a good deal of brandy per day; and she used cocaine for a year. The illness for which she was confined to the hospital began in July, 1920, when she became depressed, imagined that no one cared for her, developed ideas of persecution, thought the black hand was after her, and that she was being hypnotized. There was a certain indifference of affect; memory and intelligence seemed preserved. The pupils did not react to light, spinal fluid examination revealed a faintly positive Wassermann reaction, 10 lymphocytes to the cubic millimeter, no increase in meningeal permeability to sodium nitrite, blood Wassermann test negative. A letter written in July, 1921, by

herself indicated that she had recovered entirely and thanked the authorities for having restored her to health from such a serious illness. In this case cocaine, alcohol and syphilis had to be considered as etiologic factors; the type of psychosis added to the difficulty of diagnosis.

4. A married woman, born in 1879, who probably contracted syphilis from her husband, aside from being somewhat nervous, seemed quite well until 1914. In 1915 she was admitted to the hospital because of depression, threats of suicide, visual hallucinations, and delusions of persecution. She was disoriented as to time and place. Later she became euphoric and erotic. The pupils were irregular, reacted poorly to light but well to convergence. The first lumbar puncture gave: positive Noguchi, Lange and Wassermann tests; normal lymphocyte count. A second puncture in May, 1918, showed: 0.5 per cent. albumin; a faintly positive Nonne test; positive Noguchi and Wassermann reactions; no cells. She later became incoherent, was indifferent, laughed without cause, was subject to violent spells of anger without reason, hallucinated, and became stereotyped. A third lumbar puncture in August, 1922, was negative throughout; the Wassermann reaction of the blood was negative. In this patient there was a catatonic state with negativism, indifference, stereotypy, and dissociation of ideas. In 1918 the spinal fluid indicated syphilis; in 1922 it was normal.

5. A woman, born in 1879, addicted to alcohol, whisky, absinthe, and wine, in 1911 developed ideas of persecution, became extremely jealous and later taciturn and negativistic. She had auditory and visual hallucinations; for three years she menaced her husband, her children, and a cousin, with whom she said her husband had intimate relations; accused her husband of defloration of his daughter. She was admitted to the asylum in 1914, at which time she was oriented and seemed to be lucid; answered questions slowly but evasively and was negativistic. There was anisocoria, paresis of pupillary reactions, patellar reflexes greatly diminished. Lumbar puncture showed: a slightly positive Noguchi test, positive Wassermann reaction, no lymphocytes. She was returned to her family in 1914, after which the difficulty again appeared; she was recommitted to the hospital in 1915 because she wanted to put out the eyes of her husband. A second lumbar puncture gave the same results as the first. The third puncture fluid was negative throughout, and the Wassermann reaction of the blood was negative.

Redalié calls attention to the fact that in all of these cases syphilis was undoubtedly present and that the clinical picture was extremely varied. Further than this he permits the reader to draw his own conclusions.

WOLTMAN, Rochester, Minn.

STUDIES OF CEREBROSPINAL FLUID AND BLOOD OF SYPHILITIC AND NORMAL PERSONS, WITH SPECIAL REFERENCE TO THE IMMUNITY REACTIONS AND THE COLLOIDAL GOLD TEST ON THE ORIGINAL AND UNFILTERED FLUIDS AND SERUMS.

CHARLES E. NIXON and KOICHI NAITO, Arch. Int. Med. **30**:182 (Aug.) 1922.

The first part of this extensive research is concerned primarily with the nature of the substance in the serum responsible for a positive Wassermann reaction. The globulin and albumin were separated from the serum: globulin was precipitated with ammonium sulphate solution and purified by dialysis; the albumin fraction was filtered and dialyzed in the same way. The Wassermann reaction on albumin from syphilitic serums was negative in all instances;

whereas the reaction on globulin was positive in all instances with a marked tendency to an increase in the positivity. The globulin from normal serums gave a positive reaction in 72 per cent. of twenty-one cases. The authors conclude that the globulin fraction in syphilitic serums contained the active substance in the Wassermann reaction. Experiments on the filtrability of globulin in syphilitic serum by the ultrafilter showed that the filtrability was less than normal serum. Three factors are advanced to explain the last observation: (1) the possible ease of adsorption of the active globulin of the ultrafilter as compared with normal globulin; (2) the possible greater size of the particles of active globulin as compared with normal globulin, and (3) the possible instability of the active globulin as compared with normal globulin.

The second part of this research deals with the nature of the colloidal gold test. Globulin prepared from normal human blood serum gave distinct curves, the reduction usually occurring in Zone I; curves from globulin from syphilitic serums were somewhat more marked. The albumin fraction from normal and syphilitic serums may cause a reduction, the curve occurring in the moderate dilutions. Dialyzed syphilitic and non-syphilitic serums gave curves tending to be irregular. Colloidal gold reactions on the original and ultrafiltered serums gave fairly similar curves with a tendency to a greater difference between the zones of reduction in the syphilitic cases. The globulin: albumin ratio was found not to be a determining factor in the gold curve. Ultrafiltration of the spinal fluid was found to lessen the intensity of the curve and also to change the zone of reduction. Addition of salt solution tended to increase the intensity of the curve. Albumin tended to diminish the curve and change the zone of reduction.

The authors conclude that there are present in the cerebrospinal fluid and blood certain substances which act in a protective way against the coagulation of colloidal gold by an electrolyte; that both precipitating and protecting substances are present in pathologic fluid and the curves in Zones I, II, and III are due to varying amounts and proportions of the precipitating and protective substance; that the protective substance is decreased by ultrafiltration to a greater degree than the precipitating substance; that the precipitating and protecting powers are modified by the state of the protein; and that the salt solution used in the test partially neutralizes the protective action.

VONDERAHE, Philadelphia.

THE VIBRATING SENSATION IN DISEASES OF THE NERVOUS SYSTEM. R. T. WILLIAMSON, Am. J. M. Sc. **164**:715 (Nov.) 1922.

This article is a report of the use of a large vibrating tuning fork in the study of nervous diseases extending over a period of seventeen years. Many obscure forms of early organic disease of the nervous system are clearly differentiated and, in the author's estimation, the loss of the vibratory sense takes the same place in the sensory system as the Babinski sign in the organic changes of the pyramidal tracts.

Normally, the vibrating sensation is felt when the foot of the vibrating tuning fork is placed over the bones of the limbs, on the nails, on the iliac spines, sacrum, vertebral spines and bones of the thorax. It is particularly well felt over the sternum. On the bones of the skull, the vibrations are not felt, but are heard. On the anterior abdominal surface, at the level of the umbilicus, it is distinctly felt under normal conditions. It is also well per-

ceived between the umbilicus and the thorax. Below the umbilicus it may be felt only when firm pressure is used, or, in cases of very stout persons, it may not be felt at all.

Loss of the vibratory sense at any of the above points distinctly indicates organic changes in the sensory nervous system. When associated with other symptoms it may be considered as a positive sign of an organic lesion, and it may be the earliest sign of such a process.

A study of spinal lesions has shown that the nerve fibers conducting this sensation apparently do not decussate in the spinal cord. They are found to ascend in the posterior columns. In cerebral disease the vibrating sensation is lost along with other sensations. Its return is coincident with the return of other forms of sensation. It has no known cortical center.

The loss of the vibrating sensation may be present in the earliest stage of the disease when other symptoms and signs are very few or slight; such as in the earliest stages of a peripheral neuritis, tabes dorsalis or posterolateral sclerosis. It is one of the first indications of sensory affections in many lesions of the spinal cord, and later, loss of tactile, pain and temperature sensations may be found. It is a very delicate test and should be used routinely. In spinal anesthesia the vibrating sensation is often the first to be affected and the last to recover; while in cerebral anesthesia the vibrating sensation may be the least affected, and if affected it may recover before tactile anesthesia. Such motor diseases as anterior poliomyelitis or amyotrophic lateral sclerosis, may be excluded if the vibrating sensation is found to be lost. A paraplegia due to organic disease may be easily differentiated from a paraplegia due to hysteria, functional affections or malingering.

The loss of the vibrating sensation on the abdomen may be of service in indicating the upper limit of a spinal lesion in traumatic and other spinal affections when other forms of sensation are not affected.

Vibrating sensation is usually lost in the following diseases: Multiple peripheral neuritis due to alcohol, arsenic, diabetes, etc.; acute disseminated myelitis; acute myelitis; combined posterolateral spinal degeneration or sclerosis; tabes dorsalis; syphilitic spastic spinal paralysis; compression myelitis due to caries; spinal meningeal tumor. It may be a means of determining the level of localization in spinal cord tumors or injuries and in detecting the early presence of pernicious anemia. The diseases in which the vibrating sensation is never lost are: anterior poliomyelitis (acute, subacute and chronic); amyotrophic lateral sclerosis; sciatica; primary brachial neuritis; lesions of a single peripheral nerve; hematomyelia; syringomyelia; cerebral diseases in which other forms of sensation are retained; hysteria; functional affections, neurosis; neurasthenia, and malingering.

The paper presents in detail the finer points of differentiation between these diseases.

TEMPLE FAY, Philadelphia.

TUBERCLE OF THE TEGMENTUM PONTIS—A CONTRIBUTION TO THE SYMPTOMATOLOGY OF PONTINE LESIONS—PICK'S VISIONS. H. W. STENVERS, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:221, 1922.

The patient was a shoemaker, forty years of age, who had often had attacks of coughing during childhood and at thirteen years of age had an hemoptysis followed by a five months' rest cure. Three and one-half months previous to admission he had attacks several times a day in which he felt compelled to

swallow very rapidly over a period of about three minutes. These attacks began suddenly and were accompanied by a sour taste in the throat. After nine weeks this symptom disappeared. Five weeks previously he developed a sensation of pepper in his mouth and nose, suffered from attacks of dizziness, and had difficulty in speaking due to a "swollen tongue." Three weeks later he was unable to swallow when he tried to do so voluntarily, but did well involuntarily; paresthesia in both arms and diplopia soon appeared; there was morning nausea and vomiting. The dizziness interfered with his walking, especially so when looking at a moving object. There was no headache at any time. Urination and defecation were normal; erections were maintained, however ejaculation had been absent for eight months.

At the time of the examination in April, 1921, there was dysarthria, a normal fundus, horizontal nystagmus in looking toward the right, left or upward, diplopia on looking toward the left due to weakness in the field of the left external rectus, normal corneal reflexes, slight hypalgesia over the tongue, palate, and lips, slight weakness of the left facial, inability to hear watch on the left, past pointing laterad with either hand, atrophy of the left half of the tongue with deviation toward the right on protrusion, nasal speech, hypotonia of the right arm, general cutaneous hyperpigmentations, normal reflexes throughout, some uncertainty in gait, good muscular coordination, seven lymphocytes and a faintly positive Nonne in the spinal fluid.

While in the hospital the patient was restless, sang, and complained of vertigo. He later hallucinated, was often somnolent with attacks of arrested respiration, and complained greatly of fatigue. Early in May he said that cold water was being thrown on his face, that he heard someone rapping on the bed, that he saw remarkable things going on in the adjoining room, that people were sitting on the radiator and were jumping through the walls, that the walls of the room were bent forward and moved upward. These special hallucinations were prominent and persistent. The patient died May 8 of respiratory failure.

The delirious condition at first suggested a diffuse process; however, the peculiar visual hallucinations, first described by Pick, in which he emphasized the influence of cerebellar, vestibular, and ophthalmostatic disturbances on these, suggested a lesion in the region of the fourth ventricle, particularly of the floor.

Necropsy revealed a tubercle in the tegmentum of the pons and the medial and dorsal portion of the medulla. The upper end of this tubercle lay just caudad to the abducens nucleus, and the lower end was in the plane passing through the inferior extremity of the left olive. The posterior longitudinal fasciculus was involved.

Stenvers calls attention to the early disappearance of ejaculation in the presence of normal erections. While this dissociation has been looked on as pathognomonic of epiconus lesions, it can evidently be dependent on disturbances higher up. The occurrence of ejaculation in hemorrhage of the fourth ventricle is well known. It is likely, therefore, that ejaculation proceeds from the medulla or above. According to Oppenheim, erection probably has its center in the second sacral segment while the center of ejaculation lies below this point. The writer's observation would localize this reflex tract as passing through the district lying between the abducens and the hypoglossal nuclei.

The difficulty of orientation in space in the presence of optical stimulation by moving objects is interesting. Probably the lesion of the posterior longitudinal fasciculus was responsible for this. Pick attempts to interpret the seeing

of visions through a wall as a result of diplopia. In this patient such hallucinations were present in the absence of diplopia, although there was, to be sure, a paresis of the ocular muscles. Stenvers believes that Pick's visions are of some diagnostic importance, indicating a lesion in a circumscribed portion of the floor of the fourth ventricle.

WOLTMAN, Rochester, Minn.

AFTER-CARE OF INFANTILE PARALYSIS CASES OF THE 1916 EPIDEMIC IN BROOKLYN: A Detailed Report of About Three Hundred Cases Followed Through Five Years. H. G. DUNHAM; with an Introduction by THOMAS J. RILEY, J. A. M. A. **80**:224 (Jan. 27) 1923.

This is a report of the repeated examination of 297 patients chosen from the thousands stricken with infantile paralysis in Brooklyn in the summer of 1916, and followed through five years. It is probable that a large proportion of the population at sometime in their life history has been afflicted by the abortive or nonparalytic form, and it is conceivable that a certain number of obscure conditions of the central nervous system may have been initiated by adhesions and obstructions resulting from such an attack, unnoticed at the time of onset because no clinical signs and symptoms were present.

Of special significance is the observation that only a small group of patients previously operated on for tonsil and adenoid conditions contracted the disease, and in the patients operated on the percentage of recoveries was very much higher than in those who had not been subjected to operation.

It was determined that persons with the disease were a source of active infection at least eight days after the acute onset, and there was little evidence that the disease was contracted from a person ill more than two weeks. It is thought that the virus is carried to the different parts of the body by the lymphatic system, and, contrary to the older studies, the disease at present must be considered a general and not a local infection, but showing a tendency to localize in the nerve and lymphoid system.

The number of cells in the cerebrospinal fluid is increased to from 20 to 100, and in some cases even as high as 500 per cubic millimeter. Polymorphonuclear cells predominate in the earlier stages of the disease, preceding the paralytic period. After paralysis is established the mononuclear lymphocyte predominates in numbers, ranging from 75 to 100 per cent.

In this group only fourteen families had more than one child ill with this disease at the same time.

Particularly during an epidemic of infantile paralysis of any size, children with obscure indispositions should be kept at rest in bed for several days, for if paralysis supervenes, it usually appears within a very short time after the initial upset, the average for this series being three days after the first manifestation of illness.

In a general way, the younger the child, the nearer does restoration of function approximate the normal status. Hydrotherapy was very generally advised for all these patients, and by the majority was faithfully carried out. When possible, the child was taught to exercise the affected muscles while in warm water, which was an advantage, the extremities being lighter in water than in the air.

In this series, the back and abdomen with both lower extremities was the combination of involvement by far most frequently found. The more severely involved upper extremities did not respond to treatment as rapidly as the equally involved lower extremities.

NIXON, San Francisco.

THE MYOCLONIAS. H. Roger, Ann. de méd. **12**:150 (Aug.) 1922.

This is a critical review of the subject of the myoclonias, embracing its historic and clinical aspects as well as its pathogeny and treatment. Concerning the pathogeny, the theory of a myopathic origin rests only on necropsies showing alterations in muscles. Roger is certain such muscle changes are secondary and not primitive. The neuritic origin, defended by Manquat and Grassat, was based on the coexistence of paramyoclonus and neurofibromatosis with abolition of the reflexes in several cases. But Vanlair experimenting in a dog with myoclonus did not find the latter suppressed by curare, a poison of nerve terminals. At the present time, therefore, the discussion limits itself to the spinal theory as opposed to the cerebral.

Friedreich related paramyoclonus to an exaggerated excitability of the spinal anterior horns. Points of evidence for this conception given by Friedreich were: the symmetry of the movements; the great influence of reflex cutaneous excitations; and the abolition of the movements by voluntary movements. The presence of fibrillary contractions in both the poliomyelitides and the myoclonias is another argument. The author cites two cases, both with myoclonus localized in the legs. This localization, with the coexistence of sphincteric troubles, and bilateral positive Babinski sign, are in favor of a spinal origin of the movements.

The chief sponsor for the cerebral theory is Raymond. He incriminates cerebral lesions and points out the association of epileptic crises with certain myoclonic syndromes, and also cites the findings of several necropsies: a sclerosis in the rolandic zone in an epileptic case with myoclonus (Jacquin and Marchand), and lesions, predominantly cortical, in a case of acute paramyoclonus (Murri).

There is a tendency to incriminate the mesencephalon and in particular the corpus striatum by reason of the known localization in these centers of a great number of automatic movements. Roger writes, "Although the majority of the present facts in regard to encephalitic myoclonias are in favor of the striate origin, the spinal hypothesis cannot as yet be rejected. The number of necropsies with complete histopathology is not sufficient to settle the problem." Roger shows a disposition, to ask, with Hunt, if two localizations are not possible. Hunt has suggested that rhythmic myoclonias relate to a disordered infrastriate mechanism, and that non-rhythmic myoclonias (paramyoclonus) relate to lesions of the cord or medulla.

Davis, New York.

THE INFLUENCE OF THE NERVOUS SYSTEM ON THE DEVELOPMENT AND REGENERATION OF MUSCLES AND INTEGUMENT IN INSECTS. Stefen Kopec, J. Exper. Zool. **37**:15, 1923.

The fifth abdominal ganglions of caterpillars of *Lymantria dispar L.*, were removed after the second moult. The animals developed normally and after three to five weeks they were preserved and studied in sections. The muscles of the caterpillars did not undergo either atrophy or degeneration after removal of their corresponding ganglions. All three thoracic nerve ganglions were removed from caterpillars a few days before pupation and the behavior of the muscles was studied in the emerging moths. In the moths muscles were found only when the larval ganglion of the corresponding segment had been left intact. On the contrary, the adipose cells of the caterpillars underwent metamorphosis notwithstanding the absence of the corresponding ganglions. The hypertrophic growth of the tracheas and of the intestine in imaginal seg-

ments deprived of muscles, points to mechanical correlations regulating the normal size of the organs. In general the nerve ganglions do not influence the normal development of the hypodermis of the full-grown insect, nor that of its chitinous integument together with the wings, legs, and feelers. The fourth abdominal, segmental extremities together with their corresponding ganglions and nerves were removed from caterpillars after their last moult. The abdominal legs regenerated quite independently of the nervous system as well in regard to their macroscopic as to their histologic structure.

WYMAN, Boston.

A CONTRIBUTION TO THE PSYCHOPATHOLOGIC AND LOCALIZING VALUE OF PARALLELISM BETWEEN PSYCHIC AND MOTOR ACTIVITY. H. STECK, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:208, 1922.

The writer begins his article with the conclusion that in normal as well as in pathologic conditions there exists a parallelism between motor and psychic activity. This is seen normally in the fact that walking aids thinking. In two cases of epilepsy observed by Steck, verbal perseveration went hand in hand with motor perseveration. In the catatonic stupors this parallelism is evident; all initiative is lacking in both motor and psychic fields, although it is well known that the patient is able to register his sensations. The most striking condition in which this phenomenon is observed is in post-encephalitic parkinsonian states. In all of these cases automatic movements were accompanied by an apathy and a loss of interest apparent to both patient and relative. As far as the investigations of this condition give us an insight into it, there is seen poverty and slowness of thought and action. It is worthy of note that in some of these patients a notable lack of energy constituted a character weakness, present even before the onset of the encephalitis; it is conceivable that this poverty of activity may be bound to a constitutional weakness of the striate apparatus, which thus would constitute a locus of lessened resistance susceptible to exogenous intoxicants. In contrast to these akinetic forms, the hyperkinesias, such as the choreas, athetoses, and tics, are accompanied by lability of effect, irritability, hypervigility, and hypotenacity of attention.

Dupré spoke of the correlation of oligophrenia with motor debility; Joffroy coined the term "myopsychies" to designate this sympathy of motor and psychic functions. Motor activity is thus a mirror picture of the psychic processes, and the reverse. It is true that there are exceptions to this rule, just as we find exceptions in the mixed states of manic-depressive insanity. Thus, irritability in a case of Parkinson's disease does not necessarily contradict this thesis. Necropsy findings in these encephalitics show involvement of the basal ganglions, particularly of the corpus striatum. In some cases there were also marked lesions of the frontal lobes.

Steck concludes that one may expect to find involvement of the frontostriate system whenever a pathologic psychomotor parallelism can be demonstrated.

WOLTMAN, Rochester, Minn.

COMPARATIVE CLINICAL OBSERVATIONS ON THE INVOLVEMENT OF THE NERVOUS SYSTEM IN VARIOUS PHASES OF SYPHILIS. JOHN H. STOKES and ALBERT R. MCFARLAND, Am. J. Syphilis **6**:169 (April) 1922.

The authors studied the results of spinal fluid examination in four groups of syphilitic patients: early and late secondary syphilis, one group from the

Mayo Clinic, Section of Internal Medicine, and another group from a consultant practice. They conclude that treatment of neurosyphilis in the first two years gives a marked response; that a rise in a cell count of the cerebrospinal fluid is the earliest evidence of a meningeal reaction; increased globulin, and finally positive Wassermann reaction follow; while symptoms and signs are regarded as late rather than early signs of involvement of the central nervous system. Spinal puncture should not be made, in their opinion, until one or two injections of arsphenamin have been given; but the routine examination of spinal fluid is advised in all cases of early syphilis. Neurosyphilitic involvement takes place early; if the spinal fluid is normal during the early secondary period there is a distinct immunity toward subsequent involvement, 40 to 50 per cent. of such patients having normal spinal fluid at all stages. The vital period of the disease which offers the most favorable opportunity for preventing involvement of the central nervous system is in the first two years. In a group of 16 to 18 per cent., which includes both early untreated and late syphilis, both blood and spinal fluid appear normal. A proportion of patients varying from 5 to 8 per cent. in early syphilis, and from 14 to 41 per cent. in late syphilis, will present negative blood reactions but abnormal spinal fluids. The authors emphasize the importance of spinal fluid examination. The Wassermann reaction in syphilitics was found to be 50 per cent. positive on blood as compared with 60 to 70 per cent. positive on spinal fluid.

VONDERAHE, Philadelphia.

A NEW CONTRIBUTION TO THE STUDY OF THYMIC DEATH.

A. PULAWSKI, Rev. de méd. **39**:495 (Aug.-Sept.) 1922.

A young woman subject to rather prolonged and severe hysterical attacks was first seen several hours after the onset of one of them. She was unconscious, cyanotic, showed a feeble pulse, had irregular respiration, not of the true Cheyne-Stokes type, but "recalling rather the 'suffocation' of hysterics." Although she did not have myosis, but because of a faint suspicion that she had taken morphin with suicidal intent, she was treated actively by gastric lavage, injections of atropin, camphor, caffein, and artificial respiration. Later under the application of faradism, she temporarily regained consciousness and addressed two of the persons near her. Then she relapsed into apparent unconsciousness, and only a few hours later, while still in a profound and calm slumber, without any convulsion, having a respiration which had become regular, she died.

In his discussion of the question, "Can death be due to an hysterical attack?" the author brings forward the necropsy findings in the case. The patient presented a status thymico-lymphaticus. The thymus weighed 47 gm. The author believes that the death was in reality a "thymic death" and recalls that a moderately large group of persons who display the thymico-lymphatic condition are menaced either casually or after an insignificant accident, by sudden death.

Contrary to the former experience of the author, he found a large hyperplastic suprarenal medulla present. Another unusual feature was an outstanding "hypertrophy of the brain," so that it appeared that the brain lacked sufficient space. Such a feature has itself been claimed as a cause of sudden death. (Neusser, Hornowski, Srzywo-Dombrovoski.)

DAVIS, New York.

SPASMODIC FORCED RESPIRATION AS A SEQUEL OF EPIDEMIC ENCEPHALITIS. Irving H. Pardee, J. A. M. A. **80**:178 (Jan. 20) 1923.

The author describes certain disturbances of the respiratory mechanism that have come under his observation as a sequel to epidemic encephalitis, which have resulted clinically in disorders of breathing of a very bizarre nature. He has noted the type described by Aronson in which the respiratory rate is over forty and the effort loud and labored, suggesting the panting of a dog. In five other cases there was a type of disturbance in which intervals of normal breathing were interspersed with occasional outbursts of abnormal respiration. Without any premonitory feelings other than, perhaps, slight dizziness, the patients complain that they feel compelled to take a deeper breath; that something is compressing the lower chest which interferes with adequate depth of breathing. The breathing, therefore, suddenly becomes deeper and more rapid, usually between forty and forty-five a minute. The effort is marked, inspiration being voluntarily long and deep, with all accessory muscles called into play. Expiration is also voluntary and labored, frequently ending with a noticeable grunt. During these spasmodic paroxysms, the mental attitude is obviously one of distress and anxiety, suggesting that which is sometimes seen in vagal attacks, and is concentrated on the difficult breathing. Conversation is avoided, and, if the breathing is voluntarily interrupted, the succeeding respirations appear to be more violent, as though to compensate.

Nixon, San Francisco.

STUDIES ON THE RETINA. PHOTOMECHANICAL RESPONSES IN THE RETINA OF EREMIAS ARGUS. S. R. Detwiler, J. Exper. Zool. **37**:89, 1923.

Specimens of *Eremias argus,* a diurnal lizard common in the vicinity of Peking, China, were kept under varying conditions of light or darkness following which the eyes were prepared for histologic examination. Studies of the retina brought out the following facts: The retina of *Eremias argus* possesses single and double cones and is entirely devoid of rods, resembling in this respect that of other diurnal saurians. It is also characterized by the possession of a slightly developed fovea, situated in the center of a prominent "area." It also contains a prominently developed pecten of conical shape which is highly vascular and deeply pigmented. The foveal cones are much more numerous and much more slender than those in the extrafoveal region. They exhibit, however, all of the characteristics of typical cones in possessing conical outer segments, oil drops and paraboloids. Illumination of the eye by diffuse daylight brings about a slight contraction of the cones and a migration of the pigment. The extent of the cone contraction averages 2.3 microns in the foveal region and 2.9 microns in the extrafoveal region. The pigment migration averages 4.1 microns at the fovea and 3.4 microns in the extrafoveal region. The photomechanical responses observed in this eye, as well as those obtained from a study of the eyes of the turtle, *Chrysemys picta,* and the lizard, *Sceloporus undulatus,* have a distinct bearing on the question of the functional significance of pigment migration and changes in the visual cells in light and dark adaptation.

Wyman, Boston.

ASTHENIA AND MANIA FROM ANTITYPHOID VACCINATION. R. Benon, Rev. de méd. **39**:585 (Oct.-Nov.) 1922.

The unusualness of the viewpoint gives this report interest. The author reports that an antityphoid vaccination brought about in a patient marked

asthenic symptoms; then a manic phenomenon and for over five years he has continued in a psychotic condition, difficult of diagnosis with alternate periods of excitement and of depression, and at times great inaffectivity and even stereotypy.

The author believes that asthenia (amyosthenia and anideation) so common, if not constant, after infectious states, can be instituted also by antityphoid vaccination. These are exceptional cases which are in relation with individual predispositions. He does not regard them as contraindications for such vaccination.

DAVIS, New York.

THE VALUE OF NEUROLOGICAL EXAMINATION IN SYPHILIS. RANDAL HOYT, Am. J. Syphilis **6**:273 (April) 1922.

Hoyt calls attention to the observation that many early cases of neurosyphilis can be cured, and suggests that a very complete neurological examination be made and repeated at intervals. Cases studied by Hoyt often showed only slight neurological manifestations such as hypesthesia of feet and ankles and slight ocular affections, these being manifested as isolated symptoms and of such a character as to be overlooked unless examination is very thorough.

VONDERAHE, Philadelphia.

DYSTHYROIDISM, ANAPHYLAXIS, AND EPILEPSY. By V. M. BUSCAINO, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:261, 1922.

In 1915 Buscaino stated his belief that idiopathic epileptiform seizures represent an anaphylactic crisis which is caused by penetration into the blood stream of abnormal thyroid products. These abnormal proteins appear in the form of octahedral crystals which may be demonstrated in the thyroid by staining frozen sections, fixed in ten per cent. formaldehyd solution, U. S. P., in a saturated solution of nile blue sulphate for eighteen hours; differentiate in a 2 to 3 per cent. solution of acetic acid; wash in distilled water, and embed in glycerin. These crystals are insoluble in water, alcohol, acetone, sulphuric ether, mineral acids, acetic acid, and alcohol; they do not stain with sudan III, are not inorganic, and do not consist of fats or carbohydrates. The xanthoproteic reactions and the reactions of Millon, Liebermann-Würster, and Axenfeld are positive. The crystals are, therefore, protein. They are usually found in the colloidal material, sometimes singly, sometimes in great numbers.

The crystals are definitely related to epilepsy. In all 396 thyroids were investigated. The thyroids of epileptics or other patients having epileptiform seizures revealed the crystals in 71 per cent.; in the thyroids of nonepileptics they were found in 14 per cent. In addition to cases of epilepsy, they may be found in progressive paralysis, arteriosclerotic dementia, idiocy and mental deficiency, brain tumors, uremia, alcoholism, and were found in one case of cysticercus cyst. These abnormal proteins enter the blood stream from the thyroid and produce an anaphylactic crisis against which the body reacts by the formation of specific reagents.

Crile has removed one adrenal and three fourths of the thyroid gland and sectioned the cervical sympathetic in 11 cases of epilepsy with a good effect on the severity of the attacks and the length of the interparoxysmal period. The writer draws a therapeutic conclusion, saying that removal of the entire

thyroid for the purpose of eliminating the source of these abnormal proteins, and the subsequent administration of thyroid preparation should produce a beneficial effect on these convulsions.

WOLTMAN, Rochester, Minn.

FURTHER FACTS REGARDING THE INFLUENCE OF FEEDING THE ANTERIOR LOBE OF HYPOPHYSIS ON THE RATE OF GROWTH AND THE SIZE OF AMBYSTOMA TIGRINUM. E. UHLENHUTH, J. Exper. Zool. **37**:101, 1923.

Metamorphosed salamanders of the species *Ambystoma tigrinum* were fed on earthworms, on beef liver and on anterior lobe of cattle hypophysis. Beef liver causes a rate of growth higher than that produced by earthworms and as high as that produced by anterior lobe. The characteristic peculiarity of anterior lobe when fed to salamanders is to maintain growth for a longer period and beyond a larger size than beef liver. Anterior lobe, among the food substances tested so far, produces the largest animals. Animals fed anterior lobe may reach a size 25.4 per cent. in excess of the size of the largest known normal animal of this species, while the largest liver-fed animal exceeds the normal maximum size of the species by only 5 per cent. Since the age limit for growth was reached in none of the animals which formed the material for this report, it seems that the cessation of growth was determined by the size up to which a given substance can make the animals grow. The effects of the anterior lobe feeding upon the final size of the salamanders are not due to quantitative differences in the food and age of the animals, but to a qualitative difference existing between anterior lobe and other food substances. For this reason the assumption that anterior lobe substance does produce real gigantism in certain salamanders, is apparently even more justified today than it was previously.

WYMAN, Boston.

SOME OCULAR PHASES OF LITTLE'S DISEASE (CONGENITAL SPASTIC RIGIDITY OF THE LIMBS). WILLIAM C. POSEY, J. A. M. A. **80**:80 (Jan. 13) 1923.

The author reports three cases of Little's disease with ocular symptoms, and discusses the types of ocular involvement found in these patients. Strabismus is the most constant and nystagmus occurs next in relative frequency varying greatly in degree and kind. Paralysis of the extra-ocular muscles and optic atrophy are rare.

NIXON, San Francisco.

ANTITYPHOID VACCINATION AND GENERALIZED SCLERODERMA. NICHOLAS and GATÉ, Bull. méd. **36**:343 (Oct.) 1922.

A diffuse scleroderma, almost completely generalized, developed rather acutely in a man aged 44 following a series of antityphoid vaccine injections. (Two previous vaccine series.) The patient presented no signs of tuberculous infection and no pathology was discoverable as a possible cause of the scleroderma. "The relation of cause and effect between the vaccination and the scleroderma appears probable." A single case of this sort merits publication because of its possible bearing on a procedure, namely antityphoid vaccination, the incontestable usefulness of which received full proof during the recent war.

DAVIS, New York.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, March 15, 1923.

F. K. Hallock, M.D., *President, in the Chair*

TWO CASES OF CEREBRAL HEMORRHAGE IN YOUNG ADULTS.

Dr. Benjamin T. Burley.

The following two cases were under my observation simultaneously in April, 1922, at the Worcester City Hospital:

Case 1.—A man, aged 18, member of a healthy family, who had been generally well except that his left eye had been injured and enucleated the year before entrance to the hospital. April 18, 1922, he noticed, while at his work as machinist, that he did not feel as well as usual and developed a severe headache. At his father's suggestion, he started to return home but was seen to stagger and fall while on his way. His father followed and took him home in an automobile. His physician was called and twice administered ¼ grain (0.015 gm.) of morphin without much effect on the headache. I saw him in consultation the next morning and ordered his removal to the Worcester City Hospital. When examined, the patient was writhing in pain from headache, particularly of the right side. He complained also of photophobia and blurred vision. The right pupil was somewhat dilated but it reacted promptly; the left eye was missing. The knee jerks were lively, the left greater than the right. There was a tendency to ankle clonus on the left and positive Babinski and Oppenheim reflexes on the left. There was moderate tâche cérébrale; anesthesia was absent except for a complete astereognosis of the left hand; coördination of movements of the left arm and leg was imperfect; and there was no nystagmus nor muscular tremor. The temperature was 98.6; pulse, 56; blood pressure, 130 systolic, 70 diastolic. The spinal fluid was sanguinolent, under moderate pressure, and contained increased albumin and globulin; the Wassermann reaction was negative. The white blood cell count was 8,800.

Increased intracranial pressure was indicated by the rapid development of choked disk in the right eye and the headache became so unbearable that it was decided to perform a right exploratory craniectomy. An area 3½ inches in diameter over the parietal and upper temporal lobe was exposed, and disclosed a non-pulsating brain with somewhat flattened convolutions and resistance in the area of the posterior central gyrus. A blunt trocar inserted in this area yielded a dark clot from the depth of ⅝ inch. Ventricular tapping also evacuated old blood clots with some hemorrhagic fluid. After a subtemporal decompression with drainage, the skull-flap and skin-flap were replaced.

Absolute relief of the headache followed the operation, vision returned on the following day so that flowers could be distinguished across the room and the patient made a slow but practically uninterrupted recovery. After two or three weeks the astereognosis and spasticity of the left side had practically disappeared. The neuroretinitis of the right eye subsided markedly and the patient returned home after one month. He later returned to work and has since remained free from cranial symptoms.

Case 2.—A single woman, aged 31, an office clerk, with a family history devoid of importance, had a fibroid removed from the uterus a year and a half before entrance to the hospital and made a rather slow recovery because of a tendency to exhaustion. Although lacking somewhat of her usual animation in recent months, she had no significant symptoms until one week before entering the hospital. One morning as she stooped over she was seized with a terrific headache over the right eye. This pain lasted for from two to three hours and was accompanied by nausea and vomiting. She received "osteopathic" treatment before entrance to the hospital. Four days after the first attack, the right eyelid began to droop; two days later she had another sudden terrific headache lasting about twelve hours. This attack was preceded by a convulsion, and was succeeded by vomiting. She was taken to the City Hospital April 20, 1922.

Examination revealed a well developed and well nourished young woman, with paralysis of the third cranial nerve on the right. There was no other cranial nerve involvement, and no sensory disturbance; a moderate tâche cérébrale was present but no incoördination and no vasomotor disturbance. Headache was severe over the right eye. The pulse was 50; temperature 98.1; white blood cell count 16,000; blood pressure, 122 systolic; 84 diastolic. The fundi at this time were normal. Lumbar puncture yielded a fluid containing old blood; albumin and globulin were three plus; smears disclosed no organisms; and cultures produced no growth. The colloidal gold reaction was 1,111,231,100. After four days, the headache still persisted. Lumbar puncture was again done and yielded blood-stained fluid under moderate pressure. There was some relief from this puncture; the pulse rose from 50 to 60; and the white cell count was now 14,200. After being in the hospital one week, optic neuritis began to appear in the right eye and, a little later, in the left.

April 30, ten days after entrance to the hospital, the patient again had sudden terrific pain over the right eye, with a convulsion lasting eight minutes. Two hours later lumbar puncture was done and 25 c.c. of bright, fresh, deeply blood stained fluid was removed with some relief of symptoms.

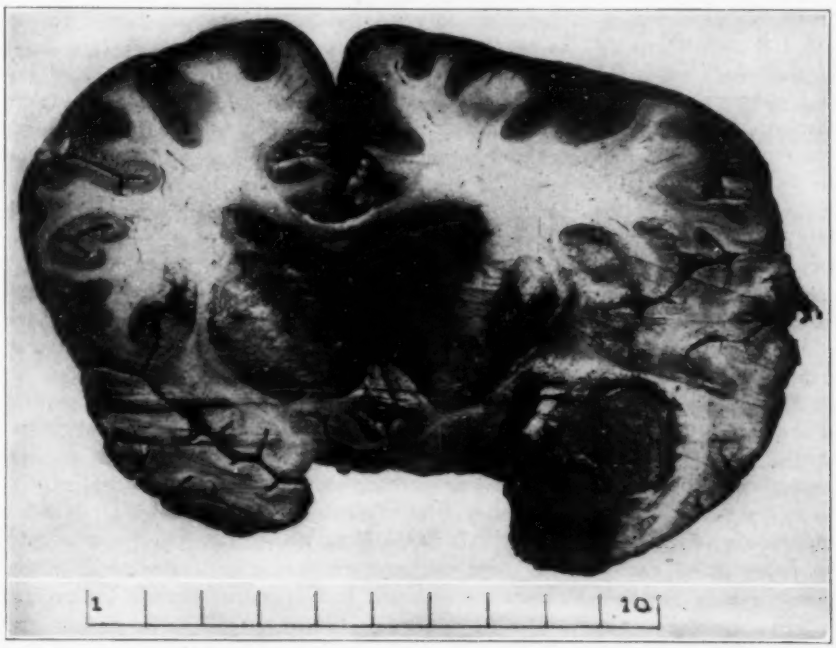
May 6, 1922, the patient was seen in consultation by Dr. E. W. Taylor. She then had definite edema of the disks of both eyes, four diopters of swelling in the right and two in the left, with some hemorrhagic areas that were more marked on the right. Lumbar puncture was again done, the fluid at this time being less bloodtinged than at the time of the convulsion. The presence of choked disks and failure to relieve the headache led to the decision to do a decompressive craniectomy on May 12. An opening two and a half by two inches over the right temporal lobe exposed a brain with a thin layer of blood clot and edema, non-pulsating and with flattened convolutions. Decompression only was attempted. The patient experienced some relief of her headache from the operation but her general condition was not improved. May 17, she had a series of generalized convulsions in one of which she died.

Necropsy disclosed a hemorrhage extending from the tip of the right temporal lobe backward and inward, communicating with the lateral ventricle and filling the ventricular system throughout. The brain was hardened in formaldehyd solution and sent to Dr. Taylor for pathologic examination.

PATHOLOGIC EXAMINATION BY DR. E. W. TAYLOR

Macroscopic Appearances.—There were present: Extensive subpial hemorrhage on the mesial and inferior aspects of the right temporal lobe, also in

the region of the cisterna magna of the oblongata, especially on the left side, extending over the cerebellum in the median line and also somewhat laterad; no hemorrhage in the interpeduncular space; considerable collection of blood in the cerebellopontine angle, especially on the left side; the third nerve on the right more or less compressed by hemorrhage. On section of the brain through the anterior portion of the temporal lobes, a large hemorrhage about 3.5 x 2.5 cm. in size was found occupying the tip of the lobe on the right, reaching the surface inferiorly and mesially. This hemorrhage had ruptured into the descending horn of the right lateral ventricle, which was filled with blood. At this point there was much less blood in the left ventricle; sections posteriorly showed both lateral ventricles and the third ventricle completely



Section through the anterior part of the temporal lobes in Case 2.

filled with blood; frontal section through the oblongata and cerebellum showed the fourth ventricle to be completely filled also. The course of the hemorrhage was evidently from the original focus in the right temporal lobe through the descending horn of the right lateral ventricle and thence through the other ventricles, appearing finally in the cisterna magna, and thence extending somewhat over the cerebellum and in the cerebellopontine angles.

Microscopic examination shows the brain tissue destroyed and invaded by hemorrhage, without evidence of tumor. Sections made through the temporal lobe in the neighborhood of the primary hemorrhage and through the region of the third nerve nucleus do not show characteristic lesions of encephalitis and although this diagnosis cannot therefore be substantiated, it is possible that a more complete examination of other portions of the brain might lead to a different conclusion.

DISCUSSION

DR. B. T. BURLEY: These cases are of interest because of the presence of massive cerebral hemorrhage in two young people; both had unbearable headaches and increasing intracranial pressure with rapidly developing choked disks, which required operative interference. Neither case showed evidence of a neoplastic or inflammatory cause. I am inclined to the opinion, however, that an encephalitic virus was a prominent factor in the etiology, this deduction being reached from my experience with three epidemics of encephalitis that followed closely after epidemics of influenza; and, further, evidence of hemorrhagic disease of the new-born appearing in the Worcester Hospitals coincident with the encephalitis. Further data of a predisposition to hemorrhage following influenza and encephalitis will be awaited with interest.

DR. E. W. TAYLOR: As Dr. Burley says, he was good enough to ask me to see his second case in consultation. The evidence of tumor seemed to me, and I think also to him, to be slight in consideration of the very rapid onset of the symptoms and the general appearance of the patient, in spite of the fact that she had a rapidly increasing choked disk with ultimate complete paralysis of the third nerve on one side. The possibility of a latent glioma with sudden hemorrhage leading to increased pressure was considered. One of the striking conditions on the clinical side was the blood-stained spinal fluid after most of the punctures, with a tendency to become deeper as the fluid was withdrawn. The massive ventricular hemorrhage must have occurred very shortly before death, in spite of the fact that the blood in the fluid would seem to point to the escape of blood into the spinal canal earlier. The microscopic examination of the area in the temporal lobe showed no tumor. There is a certain amount of reaction around the hemorrhage, which would occur in any case of such an extensive lesion.

The conclusion which Dr. Burley has reached from the clinical observation of the case is that we have to do with an encephalitis and, as he also suggests, hardly of the epidemic form. Certainly on the clinical side it is a very unusual development of encephalitis lethargica. It is possible that, when further sections are made, we may find evidence as indicated by Dr. Cobb's preliminary observations, of perivascular infiltration, which will bring it into the category of encephalitis. In the literature very small mention is made of hemorrhage as at all a constant pathologic finding in encephalitis lethargica.

Just a word about the first case, which appears to me still more mysterious. I happened to see this man also, when he was on the high road to recovery. It is difficult to see why he recovered through a simple decompression operation with the relief of his symptoms of intracranial pressure and without residuals of any sort. The cases taken together form a small group of hemorrhagic disease of unusual rarity, and it seems to me of very great interest.

DR. GILBERT HORRAX: The two cases are certainly of extraordinary interest, as Dr. Taylor has said. I was struck in the report of the second case with the fact that this woman had a complete ptosis and pupillary changes of the third nerve, and it occurred to me that we might be dealing with an aneurysm of the base that had suddenly burst. We have had several cases at the Brigham Hospital with similar neurologic signs. However, an examination was made at the base of the brain in this woman and no aneurysm demonstrated. In the first case it is a little problematic as to just what the boy had: whether the clots that came through the needle were due to trauma to the cerebral vessels or from a large clot. The lack of brain pulsation with marked tension

at the time of operation would indicate the latter. The fact of the blood filling up the ventricular system early does not seem to me at all inconsistent. The natural assumption is that such filling up would lead to sudden death, but sometimes it may go on for a long time, through the ventricles and down the cord, before causing death.

DR. STANLEY COBB: The specimens I looked over this morning indicated an encephalitis, not of the lethargic type.

DR. H. R. VIETS: I should like to ask if these cases are not somewhat analogous to the cases of spinal cord hemorrhage coming on acutely which Dr. Burley reported several years ago: acute ascending myelitis.

DR. B. T. BURLEY: Replying to the question of Dr. Horrax as to the need of operative interference in Case 1, I would call attention to the presence of rapidly increasing choked disk in the right eye, the left having been previously lost, as sufficient reason for an exploratory craniectomy with subtemporal decompression.

Regarding the question raised by Dr. Taylor as to rapid fatality from ventricular hemorrhage, these cases, particularly Case 2, showed the presence of fresh blood from lumbar puncture consequent on fresh cerebral hemorrhage, giving us evidence that hemorrhage into the ventricular spaces is not always promptly fatal.

Replying to Dr. Viets', there is an interesting analogy in the different types of acute ascending myelitis and the different types of encephalitis, particularly those with and without hemorrhage.

AN UNUSUAL TUMOR OF THE THIRD VENTRICLE IN A CASE OF DIABETES INSIPIDUS. PERCIVAL BAILEY and B. J. REIFENSTEIN.

Anatomic specimens from a case of diabetes insipidus were presented, the history of the patient being as follows:

G. H. C., a schoolboy, aged 16, with essentially negative family and past history, was admitted to the medical service of the Peter Bent Brigham Hospital, Nov. 9, 1921, complaining of thirst and frequency of urination. The patient stated that three months previous to entry he had a sudden onset of thirst, and had since drunk about five quarts of water daily, with a corresponding increase in the amount of urine passed. He was obliged to get up three times or more each night to urinate. In addition, he was very constipated and had lost about five pounds in weight.

The boy was poorly nourished, with a diffuse brownish pigmentation all over his body. The tendon reflexes were very active, with a tendency to clonus at each ankle. The Wassermann reaction was positive in the blood; the spinal fluid was normal. Antisyphilitic treatment was instituted and continued vigorously, and the polyuria was controlled fairly well by intranasal insufflation of pituitary extract.

July 17, 1922, the patient was again admitted to the medical service complaining of persistent frontal headache which had been present for two months. For two weeks previously he had had weakness of the legs, and transitory diplopia. For the four last mornings he had vomited his breakfast. He was at this time drowsy. There was an extensive acneiform rash over the body; the reflexes were very brisk; and there was general constriction of the visual fields. He was transferred to the surgical service July 21, 1922. At that time he weighed 87 pounds. His fluid intake was 1,900 c.c. and output 1,500; the

metabolic rate was —38. The blood Wassermann reaction was still feebly positive. He was very emaciated and drowsy. Roentgen-ray examination showed the sella turcica to be normal.

The patient became rapidly more feeble and on July 29, it was noted that the pupils were absolutely fixed to light and in accommodation. The left pupil was widely dilated and there was ptosis of the left eyelid. The left plantar reflex gave a dorsal response. The next day, the right pupil was also dilated and there was a slight ptosis of the right eyelid. From that time he became very weak. No further neurologic symptoms developed. On August 19, he weighed 72 pounds and his urinary output had fallen to 400 c.c. August 21, his rectal temperature had fallen to 95 F. and August 24, he died, with a terminal hyperthermia.

At necropsy, the pineal gland was found to be about twice the normal size, and in the third ventricle was a very vascular tumor, spherical, about 4 cm. in diameter. The pituitary gland was normal. The other organs of the body, especially the kidneys, were normal.

We wish to call attention to the fact that the case was not operated on because the usual symptoms of a suprasellar tumor were absent, as were also general pressure symptoms. The location of the lesion was certain because of the diabetes insipidus. Clinically, the course of the disease simulated closely a basilar syphilitic meningitis. The histologic structure of the tumor was very unusual, but a positive opinion as to its nature and origin will have to await further investigation.

DISCUSSION

Dr. E. W. Taylor: I should like to ask what is considered to be the cause of the cessation of the diabetes insipidus—the spontaneous disappearance of that symptom.

Dr. Percival Bailey: That is a very interesting phenomenon, which I cannot explain. It is frequently seen in these patients before death. It is not likely to be solved until we know much more concerning the essential mechanism of diabetes insipidus.

Dr. E. W. Taylor: Is it possible that the growth of the tumor caused the cessation of the diabetes insipidus, since, as the tumor increased, the diabetes diminished? Was it a sufficiently destructive lesion to lead to the cessation of the extreme urinary output?

Dr. Percival Bailey: It is difficult to answer these questions because of the inherent difficulty of determining whether any neurologic symptom is due to an irritation or to the removal of an inhibition. In this case the tumor had invaded the base of the brain along the perivascular spaces of Virchow-Robin and had also invaded the meninges around the cerebellum and the pineal gland. It may be that this increasing invasion led to a cessation of the polyuria. I have noticed in the case of dogs who have had a very destructive lesion of the hypothalamus that as long as they are lethargic the polyuria is absent and only appears as they gradually recover from the lethargic condition; if the lesion is less destructive the polyuria appears immediately.

FACTS AND FANCIES OF MENTAL HYGIENE. Dr. George V. Pratt by invitation.

Misinformation and confusion as to what the mental hygiene organizations are endeavoring to do sometimes appears in medical groups as well as among the laity. As perhaps the latest of the nation's great public health movements,

mental hygiene is just commencing to find its feet. It is neither a complete nor an exact science like bridge building or analytic chemistry. Its principles are not as yet sufficiently developed to permit of predicting accurately the degree and kind of response to stresses, strains, or emotional situations. It is not a kind of sect or fad or a new cult. It does not advocate a new faith or system of treatment similar to osteopathy or the electronic reaction of Abrams. It has no special technic or definite system of therapy and is not held out as a panacea for all the mental ills of the community. Instead, mental hygiene feels that, like psychiatry, it possesses a little knowledge concerning the nature, cause, and more especially, the prevention of some, not all, mental disorders. It believes this knowledge, admittedly incomplete, is nevertheless sufficient to warrant its withdrawal from laboratory seclusion and its application to actual community problems.

Mental hygiene, if it ever was, is not now in the closely guarded control of a few persons. Today mental hygiene embraces not only the science of medicine, but its practical application brings it into close touch with almost every phase of human endeavor. Thus it contacts fields of sociology, economics, criminology, and so on. Mental hygiene as a public health movement does not endorse unreservedly, extreme viewpoints on either psychanalysis or psychology. It finds value in the products of both of these schools of thought, but only as adjuncts to a program of treatment, and not the entire program itself. State Mental Hygiene Societies have widely different programs. Some engage in clinical work almost entirely, while others devote their activities to educational work. In Massachusetts where an elaborate state hospital system of out-patient clinics exists, the Mental Hygiene Society engages exclusively in educational work.

The Massachusetts Society for Mental Hygiene functions on the belief that if early detection of early symptoms of approaching mental disorder is a desideratum for prevention, then there is implied on the part of the public a knowledge of what these symptoms are. But until some of the fog of stigma, superstition, and ignorance that surrounds the mentally sick is dispelled, little can be hoped for in the way of patronage of out-patient clinics. Methods of education are numerous: In the Massachusetts Society for Mental Hygiene they include a Lecture Bureau Service supplying speakers; frequent public conferences; publication of simply worded literature; issuing of a Monthly Bulletin; insertion of courses in mental health training into the curriculums of normal schools, nurses' training schools, schools for social work, and the like. Recently, attention has been concentrated on conducting instruction or lecture courses of six to eight weekly lectures for such specialized groups as school teachers, social workers, and nurses. Much attention has also been given recently to focusing interest on the problems of childhood, in the formative, flexible age when good mental habits may best be instilled and faulty ones corrected.

DISCUSSION

Dr. Bronson Crothers: In writing a paper on mental hygiene, I wrote as an admitted amateur and outsider who had made an earnest attempt to understand the campaign. The question that came up for chief discussion was the wisdom of attempting to popularize the freudian and the more extreme psychologic points of view. In particular, I regretted the publication of books for mothers based on freudian theories and asked for authority for the statement that 50 per cent. of mental disease could be prevented by proper recognition and early treatment of conflicts. That statement, made in the heat of

a campaign for recognition and financial support, is less aggressively repeated in literature sent out by the Massachusetts Committee which quotes the Governor's message to the effect that 50 per cent. of mental disease is preventable. Except for these criticisms of detail I am, of course, heartily in favor of mental hygiene. The reason for writing at all was to call attention to the movement as a whole and to emphasize my own feeling that mental hygiene is not, as many feel, simply a division of psychiatric work. Obviously complete and whole-hearted support of a program which can be agreed on is essential. In order to stir up discussion on this subject it seemed worth while to present the reactions of an outsider to the fascinating and important series of articles published by the National Committee.

Dr. E. Stanley Abbot: It seems to me that mental hygiene is a part of general hygiene. Just as we try to build up the bodies of children and make them as healthy as we can, so the object of the mental hygiene movement is to understand what is necessary for the best development of the mental activities of the child. The mental hygiene movement is trying to find out what the principles are that lie back of all behavior as guided by our mental activities, and it is for that reason perhaps, because the Freudians have done more to analyze the mental factors of behavior, that we turn rather to the freudian psychology for help. Certainly academic psychology has done very little to help in this field. Mental hygiene has seen the need of studying these mental factors in behavior, and it so happens that the Freudians have done more than any other group to try to analyze the basis of behavior and of our mental reactions in relation to them. That does not mean, however, that mental hygienists have to swallow freudian psychology bob, hook, and sinker. I do not think any of the men in the movement really do so. It would be unwise to do it, but they do reach out in that direction to get what help they can because help does not seem to be coming from other directions.

Dr. Bronson Crothers: The question is whether freudianism can be propagated by elementary discussion among them.

Dr. E. Stanley Abbot: I do not think it can. I deprecate very much the entrance into alleged freudianism of half-baked laymen, clergymen, psychologists perhaps—various helpers of humanity who have no solid medical or psychologic background. I do not feel they belong to the mental hygiene movement. They see an economic opportunity in writing on those subjects. It is said that Freud's introduction is one of the best sellers, and many who are not qualified write books to take advantage of its vogue.

Dr. Percival Bailey: It is a question of fundamental importance to determine just how much of the theory and practice of medicine it is wise to teach the public. Every physician should give the matter earnest thought, especially at the present moment when the new journal, Hygeia, is being launched. The future policy of this magazine as well as of the journal of the Society for Mental Hygiene, will depend largely on the reaction of the medical men to the material published in it. The statement that we could prevent 50 per cent. of functional nervous disease in children if we could avoid the mental conflicts which gave rise to them does not shock me. I think the figure is probably too low. I am sure, also, that Janet, who cannot be accused of being a Freudian, would take no offense. The problem of determining the nature of these conflicts and the means of avoiding them is still far from solution, although great progress has been made.

DR. PRATT: Psychiatrists giving popular mental hygiene talks to lay groups are frequently asked afterwards, "What can you do for a child that does so and so?" It is sometimes difficult and not a little embarrassing to explain that it is impossible to offer blanket suggestions for the correction of specific behavior or conduct disorders in children. Unlike the case of the mother who brings her child to the physician for some well recognized physical defect and for which a treatment program can be outlined that would be equally applicable to a dozen cases presenting the same symptoms, one must first in this work discover the particular etiologic factor that produces the trouble. I mean, for example, that three children all presenting the general symptoms of "tantrums" and lack of discipline will almost certainly all have distinctly individual and widely differing reasons therefor. This is why we stress in public lectures that the treatment of mental or nervous conditions is a highly individual process and that each case is a law unto itself.

Another thing that embarrasses the progress of mental hygiene work in the community is the tremendously deep feeling of stigma that attaches to the whole subject of mental disease in the minds of a majority of the public. The dispelling of this stigma and fear is perhaps one of the most difficult immediate things confronting the mental hygiene organization. It prevents the early incipient case from receiving the prompt treatment that might abort permanent invalidism and it causes many people to hide their mentally sick as if mental disease were a disgrace. A woman wrote to my office this morning asking for information about the care at home of her mother who is destructive, badly hallucinated, and with a number of uncomfortable delusions. After admitting that the family physician advised institutional care this woman went on to say that, "Of course you understand mother is not crazy, but is only a little nervous and we would not dream of putting her away in an asylum." The alleviation and ultimately the dispelling of some of this fog of fear, superstition, and prejudice is a most important objective in the work of the Massachusetts Society for Mental Hygiene.

It is important to realize that various state mental hygiene societies have widely differing programs. Thus in Connecticut, the State Mental Hygiene Society finds its chief activity in the field of clinical work. On the other hand here in Massachusetts where there exists a widespread and splendid system of state hospital out-patient clinics, the Massachusetts Society for Mental Hygiene has sharply limited its work to the field of education and publicity. The work of the State Division of Mental Hygiene in establishing and conducting clinics, and the work of the Massachusetts Society for Mental Hygiene in performing important developmental work in a community along the lines of education, dovetail together nicely.

One thing I wish to bring out in conclusion: We have at times been mildly criticised for making the general statement that "Mental Disease is preventable; mental health is procurable." We admit that we have made this statement more or less of a public slogan and we also admit that, technically speaking, this statement is not 100 per cent. accurate. The precise amount of mental disease that can be prevented we do not know. Theoretically we all know that general paralysis is preventable through absence of syphilitic infection. I think we are also ready to admit that, so far as we know at present, dementia praecox is not preventable.

We feel, however, that the amount of positive knowledge in this matter, admittedly incomplete, is nevertheless sufficient to warrant making a start

toward public education in the maintenance of good mental health. The Mental Hygiene Society has been and continues to be interested in not offering mental hygiene as a panacea for all the nervous and mental ills of the community. We publicly stress the limitations of knowledge on this subject and are extremely anxious to avoid being linked up in the public mind with the "applied" psychologist, the commercial psychanalyst, the "new thought" exponents, and other tassels on the lunatic fringe.

Perhaps the most valuable single factor contributing to the advance of community mental hygiene has been the establishment of the habit clinics which Dr. Douglas A. Thom first instituted at the Baby Hygiene Station a year ago. Such clinics, dealing with preschool age children who are presenting early symptoms of personality defects or behavior disorders, are extremely valuable in the maintenance of good mental health, and, of course, the avoidance of later mental disorder as well.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, March 6, 1923

EDWIN G. ZABRISKIE, M.D., *President*

THE PRACTICAL BEARING ON DIAGNOSIS, OF THE PHYLOGENY AND CLASSIFICATION OF SPINAL MOTOR INTEGRATIONS.

DR. WALTER M. KRAUS.

The factors necessary to produce postures of the trunk and extremities in normal man are at least three: (1) Determination of the positions of the various parts of the trunk and extremities by means of reciprocally controlled antagonistic muscles. The endogenous spinal fibers by linking peripheral motor neurones, play the major part in this determination of postures. Examples of postures thus determined are the position in opisthotonos and of the leg in paraplegia in flexion. (2) Activation of the neurones which determine posture. This activation is brought about by afferent impulses entering the spinal cord through the posterior roots and by impulses of supraspinal origin, probably arising in large part in the motor basal ganglions, but also in masses of grey matter situated more caudad. When these impulses are permitted to be continuous and increased beyond normal, due to loss of control by superior controlling systems, spastic or hypertonic states result. When these impulses are discontinuous, rhythmic or arrhythmic movements result. (3) Maintenance of posture by the synergic control of the cerebellar labyrinthine mechanism.

Determination, activation and maintenance are separate physiologic activities and originate in separate anatomic mechanisms of the nervous system. The endogenous fibers of the motor portion of the spinal cord determine all of the postures of the trunk and extremities, except that of erect posture and that which occurs in the arm in cerebral hemiplegia. These two latter postures are most probably of supraspinal origin.

When muscles are activated by means of the action of the corticospinal tract (upper motor neurones of the pyramidal tract) on anterior horn cells, the postures determined at physiologically lower levels are destroyed.

In order to determine the endogenous spinal connections which correlate the various segments of the anterior horn cells of the spinal cord, it becomes necessary first to consider the anatomic arrangement of these cells in man as well as in lower animals. It has long been recognized that they are divisible into two large groups—one mesial and having to do with the axis, the other lateral and having to do with the extremities. The mesial group, furthermore, is divided into two subgroups, one dorsal, the other ventral, which supply the corresponding dorsal and ventral axial muscles (Tilney and Riley). The lateral parts of the anterior horn cell columns are present in the cervical and lumbosacral enlargements and have to do with the extremities. According to the best authorities, the localization of these cells is such that the most distal (ventral) muscles are controlled by the most dorsally placed cells.

The movements of the trunk are brought about by synergic contraction of the dorsal and ventral muscle masses on the two sides in various combinations. However, first, the anterior horn cells controlling either ventral or dorsal muscles on one side are linked by intersegmental and intrasegmental fibers. Further linking of these groups, one to another, dorsal with dorsal, ventral with ventral, or dorsal with ventral on either right or left sides, is also present. When the dorsal and ventral group on one side are made to contract in unison and in excess of the corresponding group on the other side, lateral movements are produced. On the other hand, when the right and left dorsal muscles are made to contract in unison in excess of the right and left ventral muscles opisthotonic reactions result; while, per contra, when the right and left ventral masses are made to contract in unison in excess of the right and left dorsal masses, emprosthotonic reactions result. This leads to the possibility of classifying some of the integrating functions of the endogenous fibers of the spinal cord as follows:

Unilateral axial	Bilateral axial
(*a*) Dorsal (right and left)	(*a*) Dorsal
(*b*) Ventral (right and left)	(*b*) Ventral
(*c*) Dorsoventral (right and left)	(*c*) Dorsoventral (opisthotonos, emprosthotonos)
(*d*)	(*d*) Dextrosinistral

An analysis of the integrative control of the extremities can best be made on the basis of the various phases of normal reflex stepping (Archives of Neurology and Psychiatry **9**:184, 1923). There are two pairs of corresponding opposite sets of activities: the simpler pair representing movements of animals with singly hinged appendages (fish) and represented in man by pendulum-like movements at the shoulder and hip; the second pair representing the phases appearing in terrestrial animals. These two latter phases have already been described by Sherrington and others as the flexor and extensor phases of reflex stepping. They are superimposed on the first pair and conceal it except at two stages. The muscles of the extremities are familiarly known to be divided into dorsal and ventral groups. In the first two phases of reflex stepping showing movements at the shoulders and hips, both the ventral and dorsal masses acting at these joints are each separately integrated by the endogenous fibers of the spinal cord. The anterior horn cells of the ventral group are made to act together as are those of the dorsal group. Further integrative activity combines the dorsal and ventral antagonistic muscles by reciprocal innervation to make coordinated movements possible. Furthermore, the limbs on the same side of the body, as well as the pairs of fore and hind

limbs, are similarly linked. This makes the following classification of integrating functions possible:

Primary

Unilateral Appendicular	Bilateral Appendicular
(*a*) Dorsal (right and left; one limb)	(*a*) Dextrosinistral (fore limbs)
(*b*) Ventral (right and left; one limb)	(*b*) Dextrosinistral (hind limbs)
(*c*) Dorsoventral (one limb)	(*c*) Dextrosinistral (all limbs)
(*d*) Dorsoventral (fore and hind limb; right or left)	(*d*) Dorsoventral (all limbs)

The third and fourth phases of reflex stepping represent an alternation, at the three great joints, of activity of dorsal and ventral muscles. During walking, the act of carrying the leg from the backward to the forward position by means of flexion, represents the third phase, while the act of support represents the fourth phase. The positions of paraplegia in flexion and paraplegia in extension also illustrate these two phases. The integrating activities of the spinal cord determining these positions may be designated as follows:

Secondary

Unilateral Appendicular	Bilateral Appendicular
(*a*) VDV (right and left; one limb)	(*a*) Dextrosinistral (fore limbs)
(*b*) DVD (right and left; one limb)	(*b*) Dextrosinistral (hind limbs)
(*c*) VDV—DVD (right and left; one limb)	(*c*) Dextrosinistral (all limbs; normal reflex stepping)
VDV—DVD (fore and hind limb; right or left)	(*d*) VDV—DVD (all limbs; decerebrate and decapitate postures)

When all four extremities are set into posture by the ventral muscles acting at the hip and shoulder, by the dorsal muscles acting at the elbow and knee, and by the ventral muscles acting at the wrist and ankle and below, the animal is in the extension position of decerebrate posture; while, when the antagonistic muscles are active, it is in the flexion position of decerebrate posture. That both of these corresponding and antagonistic positions are characteristic of decerebration has been recently pointed out by Bazett and Penfield. The evidence that these postures are spinal in origin and dependent on the activity of the endogenous fibers of the spinal cord is easily found by examination of the work of Sherrington, Von Monakow, Riddoch, and l'Hermitte in cases of complete division of the spinal cord in animals and man. Further proofs have already been given elsewhere by the speaker.

A lantern slide demonstration of the positions of the trunk and extremities in various nervous diseases illustrated the above described integrating activity of the endogenous fibers of the spinal cord in a number of different diseases of the nervous system.

DISCUSSION

Dr. Smith Ely Jelliffe: Dr. Kraus has set before us a scheme of the analysis of voluntary motor function which is becoming more insisted on. Considerations concerning walking and postures have been discussed largely as matters of general description and such discussions have lacked precise, dynamic concepts. Dr. Kraus' presentation is highly desirable in that he has shown that we must be more specific in our descriptions and state where the impulse arises and where it goes.

There are one or two points to which I would like to call attention and which have some connection with the work of Tilney and Riley as set forth in their book, which Dr. Kraus has quoted. They speak of an impulse arising within a motor cell and going to another motor cell. No impulse arises in a motor cell. Every anterior horn cell is only a part of a reflex arc. The impulse

comes from the outside, and the idea of its autonomous origin in the cell is a faulty concept. The spinal cord never exists by itself and cannot be explained on the basis of its own activities. It must be related to external environmental factors.

I do not think that the concept presented by Dr. Kraus regarding the columns is correct. We cannot insist too dogmatically on the continuity of columns, which, in reality, are more or less broken or segmental. The cell groups are arranged in columnar form, but one cannot speak of the function of a column for a column has no function, although different segments within the column have partial functions in the handling of reflex activities. My interest in the localization of muscle groups has extended to attempts at explaining the breakdown in various types of integrations in cases that are not fully understood. It may be remembered that I have expressed views on the connection between vascular integrating factors and muscle group integrating factors in multiple sclerosis of certain types, and have tried to show that unconscious wishes on the part of the individual to bring into effect certain types of activity on the environment (kicking the world in the face, for instance) gives rise to changes in the muscular and vascular integrative factors, which may determine a breakdown in parts of the machinery, related to those that Dr. Kraus has brought forward.

In connection with the subject of exposure, I cite an observation: A patient was brought to my office with numerous records setting forth that she was subject to an hysterical limp. I noticed certain affective reactions as the patient walked and I asked whether a roentgenogram had been taken and was told that it had not. When it was done it showed what appeared to be a myeloma, but this was later reported to be a giant cell sarcoma, of a non-malignant type, at the lower end of the left tibia. I took considerable interest in the genesis of the giant cell sarcoma and asked myself how this could have occurred, as I believe that all problems in medicine have a neurologic basis. I first told her husband that the difficulty was surgical.

In the process of investigating her general psychic state, it soon became apparent that she constantly held the left leg, night and day, in a continuous postural attitude. Study of the unconscious showed that this posture served two purposes: first, partially closing the vagina from attack from the external world, that is, unconsciously protecting herself from sexual assault; and second, keeping the labia together in order to produce masturbatory activity. It would be interesting to find out what muscles are involved in such a postural attitude and what relationship these muscles have with the ligaments at the lower end of the tibia concerned in the pull of the chronically used muscles. Further, what is the relationship between the chronic stimulus of the ligamentous play on the bony structures which we know are inter-related? Is it possible that a chronic postural attitude can have relation to a chronic inter-related reaction of the ligaments, producing an irritation in the underlying bone and causing a giant cell sarcoma?

DR. W. M. KRAUS (closing): It is, of course, true, as Dr. Jelliffe has remarked, that impulses do not arise within motor cells, but are dependent on impulses afferent to the motor cells. I have not discussed the relation of the extent of these columns to embryologic groups of muscles, since that is an extensive subject. The existence of a column of cells depends on the appearance in the course of evolution of a new group of muscles. The mere fact that several lateral columns of cells have been added to the mesial columns, where limb control has become necessary, indicates this. A definite column of cells

has only the functions of controlling, subservient to afferent impulses, the particular muscle it serves, and so far may be said to have a definite function.

As to the genesis of a giant cell sarcoma on the basis of postural reflex, that transcends my powers of imagination. But the muscles and ligaments involved in the production of any given posture may be described without difficulty.

REMARKS ON THE HISTOPATHOLOGY OF AMAUROTIC FAMILY IDIOCY. Dr. Joseph H. Globus.

In the course of an anatomic study of the infantile (Tay-Sachs) and juvenile (Vogt-Spielmeyer) forms of amaurotic family idiocy, the following observations were made: (1) The total or partial cell changes, as described by Shaffer, form the background for all forms of this disease. (2) These common features, as well as the occurrence of transitional forms, do not allow of the separation of the disease into anatomic groups, but permit only a division into clinical forms. (3) Contrary to the general impression and repeated statements that the disease process is limited to the gray matter and involves every ganglion cell of the cerebrospinal axis, it can be said that the white matter in many instances is equally involved and that many cells in the gray matter may escape the disease process. (4) The frequent involvement of the cerebellum must be looked on as extension of the same pathologic process and not as another added feature to the disease. (5) Each case, or group of cases, may have its peculiar staining reactions. This staining variation is simply an expression of the chemical character of each case or group of cases. (6) In the complete absence of inflammatory changes, and in the presence of all the earmarks of a degenerative lesion, it may be concluded that the disease is degenerative in character and due to some metabolic disturbance.

The article was illustrated with lantern slide demonstrations.

DISCUSSION

Dr. I. Strauss: While there are variations in the disease process, the fundamental changes are present in all cases of this kind. Those of a secondary nature may vary in intensity and distribution. In the case which I studied, and to which Dr. Globus has referred, I observed very little change in the white matter. The spinal cord showed involvement of the anterior horn cells, but no change in the fibers of the cord when stained by the Weigert-Pal method. The interest to me in this disease rests in two factors. In the first place, this variety of disease (including the forms described by Vogt, Spielmeyer and Sachs) is one of the few diseases of the central nervous system that has a characteristic histologic picture, which makes the diagnosis of the disease absolutely positive. Another point about the disease is that we are dealing with something that is very fundamental in the organism, namely, with changes in the germ plasm, as well as factors introduced of an endogenous character. It has not been discovered what it is that causes such tremendous destruction of the cellular elements. In all the cases one notices that the children are remarkably well developed infants and show no signs of paralysis. Even if they do not die because of the disease, but from intercurrent infection, one finds a very extensive disease process and one wonders how the function of these cells can persist to the high degree in which it does, in spite of the remarkable morphologic change and destruction. It points out one fact, and that is, that when there occurs what appears, morphologically, to be a very

marked alteration of the cell, still there is enough function apparently going on in that cell to maintain the processes of life. Therefore there is something in the functional activity of the cells of the central nervous system of which we know very little at the present time; and they cannot be measured by their morphology. I am associated with another worker in the study of these brains from a chemical standpoint and it may be that we shall find definite chemical changes in the constituents of the brain. That, of course, will only be another side-light on the question. I think the actual discovery of the cause of this remarkable disease is something which is going to baffle us for many years, until we know something more regarding the actual nature of the function of the central nervous system cells.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, April 3, 1923

E. G. ZABRISKIE, M.D., *President, in the Chair*

MEMORIAL TO WILLIAM M. LESZYNSKY, A.B., M.D. DR. J. RALPH JACOBY.

We greatly regret the recent death of Dr. William M. Leszynsky who was for many years intimately associated with the work of this Society, was one of its former presidents, and was always an earnest and untiring co-operator in its various activities. Dr. Leszynsky was born at Newport, Rhode Island, June 16, 1859. After leaving school he took the scientific course at the College of the City of New York, where he received the degree of Bachelor of Arts. In 1876, he entered the University of New York Medical School, and received his degree of Doctor of Medicine in 1879. At the time of his death, March 3, 1923, he was Attending Neurologist to the Lebanon Hospital and the Manhattan State Hospital, Consulting Neurologist to Chirst Hospital (Jersey City), Harlem Hospital and the Peoples' Hospital. He was for many years Consulting Neurologist to the Manhattan Eye and Ear Hospital and the Demilt Dispensary, and Lecturer on Mental and Nervous Diseases at the New York Post Graduate Medical School.

Dr. Leszynsky was a member of the New York Neurological Society, the New York Academy of Medicine, American Medical Association, American Neurological Association, Medical Society of the Greater City of New York, Society of Medical Jurisprudence, Metropolitan Medical Society, New York Physicians' Association, New York Medical Union, Harlem Medical Association, Manhattan Medical Society, Medical Society of the County of New York, New York Medico-Surgical Society, Physicians' Home, Society of the Alumni of Lebanon Hospital, Association for Research, National Committee for Mental Hygiene, Thomas Hunter Association of Grammar School No. 35, etc.

Dr. Leszynsky was a prolific writer and contributed many scientific articles to medical literature. His attainments in the field of neurology were broadly recognized. Here, in New York City, his endeavors were most appreciated, because we were able, as his associates, to come more closely in contact with his personality and to know the human side of him. In our Society we came to know him not only as a man of precise knowledge and high scientific

attainments, but also as one so modest and retiring that the very qualities that made him preeminent in his profession sank into the background of his geniality and personal charm.

The New York Neurological Society has passed a resolution that a copy of this Memorial be sent to his widow, with expressions of deepest sympathy; and that copies also be forwarded to the Journal of Nervous and Mental Disease, the Archives of Neurology and Psychiatry, and the New York Medical Journal and Medical Record.

LUMBAR ARTHRITIS AS A CAUSE OF SCIATICA. Dr. P. William Nathan (by invitation).

The symptoms of sciatica, as usually described, cannot be correlated with an actual lesion of the nerve trunk or the sacral plexus. The neurologic symptoms are evidently radicular and are more or less exactly similar to those that are known to accompany lesions of the vertebrae and the cauda equina. The spasm of the muscles, the deviation of the spine and the fact that the symptoms are aggravated by movement and alleviated by rest, strongly suggest that the lesion is of an inflammatory nature involving the lumbar vertebrae.

In an endeavor to confirm this a priori conclusion, 140 consecutive cases of so-called sciatica were examined roentgenographically at the Mount Sinai Hospital and in private practice. Of these, 126 showed more or less extensive arthritic changes in the lumbar vertebrae. It may, therefore, be concluded that in a large majority of cases with sciatic syndrome, the cause is to be sought in inflammatory vertebral lesions. It is, however, necessary and at times difficult, to exclude tuberculous and malignant diseases in this region.

DISCUSSION

Dr. C. C. Beling: In what position were the roentgenograms taken, upright, or lying down?

Dr. Nathan: They were taken lying down.

Dr. Beling: Dr. Nathan has presented a group of cases of sciatica due to vertebral disease, but some cases are due to muscular changes. In 1904, Gowers stated that some cases of sciatica were due to inflammatory conditions of the white fibrous tissue of the muscles, and thought that the spindle cells of the muscles and tendons were affected. When we speak of pain on the outer aspect of the thighs it is due to lack of differentiation of the location of the pain. Sometimes affections of the external lateral cutaneous nerve are mistaken for sciatica. Sciatica is simply a symptom-complex that may be due to many conditions, and one of the commonest causes is doubtless neuromuscular fibrositis.

Dr. Rosenberg (by invitation): I have seen many cases at the hospitals and also in my office. One thing, which I have always insisted on, is the roentgenographic examination of the hip and spine. Although many cases have been studied, the report of the roentgenologist has always been negative; in fact, I can hardly recall a case in which the vertebrae have been shown to be involved. Occasionally sacro-iliac dislocation is reported, but not one case where the vertebrae are involved. The pictures shown by Dr. Nathan indicate definite vertebral involvement. Clinically, one sees cases of so-called sciatica with definite scoliosis, which, with comparatively short or light treatment, are relieved of pain and at the same time of the scoliosis. That has

been observed many times. The patients seem to suffer from a muscular contracture that causes the scoliosis. That is why I also need to know in what position the roentgenograms were taken. I have seen scoliosis with an appreciable difference in the measurements of the hip. The orthopedic condition disappeared after an epidural injection. One is puzzled to know how the condition, which apparently is one of vertebral involvement, can be affected by an epidural injection.

DR. NATHAN: I cannot say much in regard to the first speaker's remarks. I have never been able, in the examination of a large number of patients, to demonstrate a condition which resembles so-called fibrositis. Patients often complain of pain at the origin of the muscles, and myositis is not unusual, but one is not able to demonstrate that it acts in such a universal manner as has been claimed. Dr. Rosenberg's view-point regarding recovery from sciatica is well taken. There are a considerable number of cases that come in as sciatica, having scoliosis, etc., which clear up after epidural injection. This is perfectly true. Lumbar spondylo-arthritis, like other joint conditions, may be mild, or it may come on acutely and disappear with or without treatment. In some cases the scoliosis is due simply to muscle spasm which disappears with the subsidence of the inflammation; in others it is due to bony involvement and destruction of the tissues; but even these, when treated early, are amenable to orthopedic treatment. With a well-equipped roentgen-ray laboratory, and the services of a good roentgenologist, changes may be detected in the vertebrae in many cases, although in some they are not marked. In only fourteen of 140 cases were we unable to detect any vertebral change. The point I wish to bring out is that the syndrome, sciatica, cannot be brought into correlation with a condition in the sciatic nerve trunk. The symptoms as they are described and as they are encountered in practice, are characteristic of root irritation and, as can be demonstrated, this root irritation is most often induced by lumbar or more extensive spondylo-arthritis due to nontuberculous infection. However, as malignant and other diseases in this region induce similar symptoms, these must be carefully excluded.

BARBITAL INTOXICATION. DR. IRVING J. SANDS.

This report analyzes a series of fifteen cases illustrating the various manifestations of acute and chronic barbital poisoning. The drug primarily causes dilatation of the small vessels and capillaries, producing a slowing of the circulation, reduction in oxidation, heat dissipation, and a lowering of the temperature. The slowing of the cerebral circulation and the reduction in the rate of oxygen and carbon dioxid exchange in the cerebral capillaries are responsible for the hypnotic effect of the drug. In small physiologic doses, about 90 per cent. of the drug is recoverable from the urine; in larger and in toxic doses only about 50 per cent. is found in the urine, the remainder being stored in the tissues of the body.

Acute barbital intoxication results from the imbibition of a large dose of the drug, usually over 50 grains. The individual is comatose; the temperature is subnormal or moderately elevated; the pulse is slow or rapid; and the respiration may be lowered or increased. The face is flushed, the lips and finger tips are cyanosed, and the extremities are cold and clammy. The blood pressure is low; the urine contains albumin, granular and epithelial casts, and barbital. The tongue is heavily coated and the breath foul. There may be incontinence of urine and feces. The superficial as well as the deep reflexes

are diminished, and in the more serious cases may be absent. The pupils may not react to light or in accommodation. Spontaneous contraction and dilatation of the pupils are regarded by some observers as pathognomonic of barbital intoxication. On regaining consciousness, the patient shows a thick, drawling, indistinct speech, which resembles the speech of the general paralytic. There may be diplopia and nystagmus is common. Delirium may supervene. Death usually results from bronchopneumonia. At necropsy a general hyperemia of all organs is found, with cardiac dilatation, pulmonary edema, bronchopneumonia, degeneration of the convoluted tubules of the kidneys and fatty degeneration of the liver. Except for the presence of hyperemia, the brain is normal. Barbital is recoverable from the various organs. Recovery of the individual from 125 grain doses has been frequently reported in the literature; larger doses usually prove fatal.

Chronic barbital intoxication results from repeated small doses of the drug. The patient is somewhat cyanosed, and there may be erythematous skin lesions. The station and gait are ataxic and there is a general dyssynergia. Speech is drawling and indistinct; the breath is foul, the tongue coated and constipation is present. The body temperature may be subnormal, the blood pressure lowered and the pulse slow. Mentally there is an irritable, suspicious attitude and manner, emotional instability, defect in attention and poor retention. Memory, especially for recent events, is poor. Judgment is defective, and there is poor insight into the condition. These cases must be differentiated from general paralysis, epidemic encephalitis, uremia, and cerebral neoplasm.

The personality of individuals who suffer from barbital poisoning is worthy of attention. Experience leads to the conclusion that the vast majority of persons with acute barbital intoxication belong to the manic-depressive group of people, while those with chronic barbital intoxication had the personality of constitutional psychic inferiority. Therefore one often finds that these patients take the drug with suicidal intent. Furthermore, the patients with chronic intoxication, take the drug as a flight from reality whenever faced with a situation that entails painful emotional feeling. Such people often take alcohol, and many of the chronic barbital intoxication cases occur in alcoholics. Several of these patients admitted drug addiction as well.

The limitation of the sale of barbital to physicians' prescriptions, and the use of the term diethylbarbituric acid, rather than the trade name veronal or barbital would materially limit the number of poisoning cases.

DISCUSSION

DR. S. R. LEAHY: Dr. Sands has covered the ground very well. I disagree, however, in some minor details. I do not think the coma is characteristic of this condition; all drug intoxications are similar in that respect; also, it is not always possible to distinguish the coma from that of an organic disease. These people have often been users of others drugs: bromids, phenobarbital and others. I have seen one man with delusions of jealousy against his wife and great excitement, as the result of small doses. Every effort was made to prevent toxic symptoms and there may have been a psychosis mixed with the drug intoxication. I have seen a case with bromid intoxication, but the other features were not characteristic. In this case there was a history of depression ten years previously which lasted for several months. With the clearing up of the intoxication symptoms the woman developed a typical manic attack and had to be committed. These cases of barbital intoxication are very

often diagnosed as general paralysis. The ataxic gait and reduction of the knee jerks suggest general paralysis. In every case it is necessary to study the serology. I am in hearty agreement with restricting the sale of this drug without prescription.

Dr. W. J. Dougherty: In regard to limiting the sale of barbital as suggested by Dr. Leahy, the Board of Health recently added to the Sanitary Code a clause prohibiting the sale of barbital without a physician's prescription; since then the number of cases of barbital poisoning brought to Bellevue Hospital has been very small. Prior to the enactment of this restrictive clause cases of barbital intoxication were always present on the wards of the alcoholic and psychopathic service of Bellevue; but now it is the exception to encounter cases of this type. One was received recently, the first in some weeks. This man formerly spent most of his time at Bellevue—literally going out by one door and in by another.

I do not believe that barbital addiction is to be regarded as an expression of a manic-depressive psychosis, as one seldom finds manic-depressives who are barbital addicts, although some depressed cases resort to the drug to terminate their existence. There is no doubt that barbital addicts are, in most instances, examples of constitutional psychopathic inferiority, and that such persons feel incapable of facing the ordinary vicissitudes of life; they, hence, have recourse to barbital in an effort to efface themselves from the scene for varying periods of time. They may be regarded as potential suicides, desiring to escape what seem to them the painful experiences of life, the suicide permanently, the addict usually only for the time being. Confirmation of this theory is to be found in the number of barbital addicts who actually attempt suicide.

I have seen only one case of fatal barbital poisoning. This man was found unconscious on the street with narcotics in his possession and was supposed to be suffering from morphin poisoning. His respirations were, however, too rapid for morphinism. The pulse was fairly rapid; the pupils were contracted, but not to the extreme degree observed in opium poisoning. He died half an hour after admission to the hospital; shortly before his death his respirations were but four a minute. This case did not exhibit the muscular twitchings that Dr. Sands has spoken of in fatal cases of barbital poisoning. The case went to necropsy and the laboratory findings indicated that barbital was the cause of death.

Dr. B. Onuf: I have been much interested in regard to the relation of barbital addiction to manic-depressive states. Dr. Sands finds acute barbital poisoning mostly in manic-depressives; chronic poisoning, mostly in cases of emotional instability. Now, I do not know that we can make a sharp distinction between emotional instability and the manic-depressive psychoses. I think the manic-depressive temperament which is at the bottom of the latter, has many varieties. Some of its possessors are predominantly depressives, others prevalently manics, both often within physiologic limits. In still others there is an intimate mixture or alternation of these opposite trends and it is these who show particularly what can be termed emotional instability. In my writings on the manic-depressive temperament I have put down as one of the characteristics of this temperament or constitution, that those who possess it are very apt to become addicts to habit forming drugs, sometimes, as in the case of insomnia, for their narcotic and soothing effects; sometimes, as in the case of brain fag, for their exciting and efficiency increasing effects.

When the soothing and soporific effect is desired, barbital is often preferred, for its milder action, to other narcotic or hypnotic drugs. I think this inclina-

tion to take habit producing drugs is illustrated by what we hear of conditions at Hollywood. This class of people can be described as being endowed mostly with manic-depressive temperaments; and drug addiction is said to be prevalent in that colony.

Dr. E. G. Zabriskie: I am reminded of a general remark about conditions in China. When the authorities there made a strong campaign against opium, the consumption of gin increased 1,000 per cent. If we limit the selling of barbital, what is the addict going to do next? We should consider these people as definitely of a psychopathic constitution, and attempt to guide them rather than merely restrict the sale of any particular drug.

Dr. Sands (closing): I have cases illustrating the points to which Dr. Leahy referred. Formerly, I invariably mistook these cases for general paralysis. I recall several patients whom I considered as subjects for the state hospitals, only to change my mind as soon as they disposed of the drug. In regard to Dr. Dougherty's statement, I came to the conclusion that these acute cases really did not want to die; what they really wanted was to get rid of their unhappy personality. In studying the entire "longitudinal section" of their life's reactions, it must be admitted that the acute barbital intoxication cases are really manic-depressive individuals, while the chronic barbital cases are examples of the emotionally unstable type of the constitutional psychopathic inferiority group of individuals. These patients have shown other peculiarities in conduct besides taking barbital.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, March 23, 1923

Charles M. Byrnes, M.D., *President*

APOPLECTIFORM ONSET OF LEFT TEMPORAL LOBE SYMPTOMS PROBABLY DUE TO ENCEPHALITIS. Dr. Williams B. Cadwalader.

The patient, 36 years old, is right handed, and had been entirely well until Jan. 2, 1923, when, while washing an automobile, he suddenly felt faint and weak. This was followed by a sharp pain in the left side of the forehead, after which he had to lie down and vomited repeatedly. He was immediately seen by a physician, who described his condition as one of confusion. In answering questions he repeated the word "lights," misplaced words and did not seem to comprehend. On examination the following day, he presented no physical abnormalities except that on being questioned he replied in a jargon-like manner, misplacing and misusing words, so that his speech was not understandable. (Jargon aphasia is the earliest manifestation of beginning word-deafness, and the improper use of words may precede distinct signs of word-deafness.) He could not carry out a simple command, such as to put one hand on his head or to open his mouth. He had no fever and no other evidence of intoxication or illness. There was no hemianopia.

Two days after the onset, ophthalmoscopic examination made by Dr.J. T. Carpenter showed paleness of the temporal portion of each disk. On January 19, the right optic disk was swollen 1 diopter, the left optic disk 2 diopters. At that time, I regarded this case as probably a left temporal lobe lesion, either

a tumor or encephalitis. He was transferred to the University Hospital, and the following positive signs were found: There was distinct word-blindness and he missed as many words in large type as in small type. Hearing for sounds was normal on each side, but there was distinct word-deafness. He could perform simple commands, but the execution of more complicated commands was impossible. The jargon aphasia had disappeared entirely.

January 25. Dr. de Schweinitz found: in the right optic disk 1.5 diopters of swelling; in the left optic disk 3.5 diopters of swelling with large hemorrhages on the nasal side, but no hemianopia. Blood count, spinal fluid, Wassermann reactions, and urine examinations were all negative.

January 30. The aphasia had improved and now resembled an amnesic type of asphasia. Word-deafness and word-blindness, though still present, were decidedly improved. Dr. de Schweinitz again reported the presence of bilateral choked disk, higher on the left. Spontaneous writing was impaired.

February 10. Dr. de Schweinitz reported 1.5 diopters of swelling in the right optic disk and 4 diopters of swelling in the left; this was practically the same as before, but he thought the appearance of the swelling was different from that of tumor.

February 15 and 21. Examinations by Drs. Halloway and Williams revealed that the swelling of the disk was diminishing.

March 1. Dr. Baer reported the swelling of the left disk as 1.5 diopters, and of the right disk as 1 diopter; on March 14 both disks showed about 1 diopter of swelling. Since then the swelling, as well as all other symptoms, has almost disappeared.

The case presented at first many difficulties in diagnosis. In the early part of January the patient appeared to have typical jargon aphasia, word-blindness and word-deafness, with gradually increasing bilateral choked disk; all of which began with an apoplectiform onset. The sensory aphasia gradually subsided and has disappeared. Absence of homonymous lateral hemianopia in the presence of the signs of involvement of the left first temporal convolution and angular gyrus, with decided improvement after a comparatively short time, seemed incompatible with the diagnosis of cerebral tumor.

He was treated by repeated lumbar punctures with removal of a small amount of fluid each time, and was given concentrated magnesium sulphate solution on the theory of diminishing intracranial pressure from edema. He seems to have made a complete recovery. I consider this case as probably one of encephalitis of the left temporal lobe.

DISCUSSION

Dr. William G. Spiller: The improvement in this man's eyegrounds has been most satisfactory. He permitted only two lumbar punctures, and declined after that to have any further reduction of intracranial pressure in this way. I employed concentrated magnesium sulphate solution by enema in the method suggested by Fay. The magnesium sulphate may be given by mouth or enema. The highest swelling of the disk was 4.5 diopters. Dr. Langdon examined the patient this afternoon and found that the swelling had practically disappeared. I regard this case as one of meningitis serosa or encephalitis.

Dr. J. Hendrie Lloyd: This case appears to be one of vascular obstruction; the sudden apoplectiform onset, the delimitation of the lesion to the language zone, without paralysis, hemianesthesia, or hemianopia; all these features make it look like a vascular lesion. The condition of the eye grounds, as reported, is rather puzzling, but as this has tended to clear up, it evidently does not indicate

a grave organic lesion, whatever the cause may have been. Whether encephalitis can cause such a strictly localized lesion, of such sudden onset, is an interesting question.

DR. WILLIAMS B. CADWALADER: The sudden onset of symptoms did suggest a vascular lesion, but when the choked disk developed I felt that explanation was insufficient. I do not believe the symptoms were due to a vascular lesion alone.

SIMILAR HYSTERICAL CONTRACTURES IN WIFE AND HUSBAND.

DR. T. H. WEISENBURG.

Two cases of hysterical contracture are reported because of their unusual nature and occurrence first in the wife, later being imitated by the husband. The first patient, a Russian Jewess, aged 45, had been in this country 19 years. She had had nine childen, two of whom were dead. The patient was well up to six years ago when she noticed a flexor contracture in the fifth digit of the left foot. In a short time, similar flexor contractures of all the toes of this foot developed. Approximately a year later, she noticed that the middle finger of the left hand was flexed, this being soon followed with similar contractures of all the fingers of this hand. These contractures have been present for three years.

The patient complains of constant numbness and needle-like sensation in the affected limbs. The toes are more contracted than the fingers. When her attention is directed elsewhere the contracture is relaxed. It is much easier to relax the contracture in the fingers than in the toes. When the patient is emotional or talks about her troubles, the contractures are worse. This is also true when she is observed. Sensation of all forms is disturbed in the hand and foot and impairment can be easily suggested elsewhere. Neurologic examination shows increase of reflexes and no other symptoms.

The patient is very emotional, dwelling constantly on the ingratitude of her children whom she rarely sees. Investigation by the social service department of the Orthopedic Hospital, where the patients were observed, demonstrated that the patient and her husband were in extreme poverty and were more or less dependent on the charity of the children, which was precarious. The contracture dated from the time when the children left the patient and the poverty began.

The second patient is the husband, who is 47 years of age. He had been a shoemaker and had been earning about $15 or $20 a week until three years ago when he lost his trade because of the advent of machinery, and now makes only $3 or $4 a week. The contracture is present only in the right hand, having begun about four months ago in the fourth and fifth digits, and is similar to that of his wife. It has gradually extended until it has involved the other fingers, with the exception of the thumb. In the last examination, a beginning flexor contracture was noticed in the second toe of the left foot. The contractures in the husband are not as intense as those in the wife. Sensory disturbances can be found and are altered by suggestion.

It is interesting that the contractures in both the wife and husband began at the time of greatest stress, that is in the woman when the children left her and in the husband when poverty was at its worst. In the woman, the contractures were present in the left hand and foot and of the flexor type, whereas, in the husband, while of the same character, they were in the right hand and are now extending to the left foot. There is no question that they are hysterical in character.

DISCUSSION

DR. J. HENDRIE LLOYD: Cannot this be called an hysterical contracture, an imitative psychosis? I can hardly see how else to explain it. There is a curious condition known as lock spasm, perseveration. I have seen it in a case of gross lesion of the motor region of the brain, in which the patient took a firm grip and could not let loose. But I cannot imagine a husband and wife each having such a lesion in the brain. I should think it was an imitative psychosis.

DR. A. M. ORNSTEEN: I saw this woman a year ago in the service of Dr. Bochroch at the Samaritan Hospital and she presented the same syndrome that she now shows. We believed at the time that there was a large emotional element in the case. She was always accompanied by her husband who seemed to be very much concerned about her and would watch for the spasm to develop, calling attention to it each time. From the obstetrical position of her hand it was suspected that she might have tetany and I examined the woman's muscles for the spasmophilic electrical reactions, but they were negative. Now it seems that concern and anxiety of the husband about his wife's spasms, and the close association with them has helped him to develop this identical condition purely through suggestion.

JUVENILE GENERAL PARALYSIS WITH ONSET AT SEVEN YEARS.

DR. F. H. LEAVITT.

This patient, a white child aged 8 years, was admitted to the Orthopaedic Hospital and Infirmary for Nervous Diseases, at the request of the mother, who stated that the child's entire manner of life, habits, character and actions had changed and deteriorated since seven years of age. The history of the mental deterioration (in contrast to her former active mental state), the serologic reports, typical of paresis, a family history of syphilis and two miscarriages by the mother, and the physical signs lead to a diagnosis of juvenile general paralysis with its onset at seven years. The family history is that the mother had been treated for syphilis before marriage. Her first child was syphilitic and born dead. The second child was the patient who was born at term after the mother had had antisyphilitic treatment during the entire pregnancy. The third pregnancy resulted in an abortion at three months. The mother's blood reaction was strongly positive, the father's negative.

The patient showed no evidences of syphilis at birth. She began to walk at twelve months of age and to talk at fourteen months. She developed normally into a lively, alert and energetic child, talked well, was obedient and cleanly in her habits and advanced through the second year at school. At seven years of age, her conduct and mentality began to change. She had outbursts of uncontrollable temper, screaming, kicking and endeavoring to destroy everything that came within her reach. She never had any real epileptiform attacks nor paralyses. She has demented steadily since that time, having become practically unmanageable; she pays no attention to the commands of her mother, is restless, and pays no attention to the demands of bowels or bladder and soils her clothes or bed as the case may be. She has forgotten how to dress herself and the uses of knife, fork, and spoon. She eats solely with her hands, spilling much food on herself and the table. She has become cruel and resistive to children and her pets. Her outbursts of violent screaming, fighting and anger occur at any time. She also has spells in which she jumps from bed, dances around,

makes facial grimaces, sings or cries a little, waves her arms in the air then goes back to bed, lies down and sleeps. She had measles at two years, but has had no other infections or accidents.

Physical Examination.—She is a very plump and well developed girl with demented facies. The bridge of the nose is flat. The teeth are widely separated and poorly erupted but not Hutchinsonian in type. She speaks with an indistinct, choreic "thick" voice. There are slow choreo-athetoid movements in the facial muscles and a type of choreiform incoordination in the use of both arms. Her pupils are regular in outline, equal in size, and react well in accommodation, but sluggishly to light. She walks unsteadily, with feet wide apart for a good base of support. The knee jerks are very hyperactive but equal; there are no ankle or patellar clonus and no spastic symptoms. Visceral examination is grossly negative. The Wassermann examination of the blood and spinal fluid showed plus 4 with all antigens; the spinal fluid cell count was 166 and the colloidal gold curve was 5,555,432,210.

GUNSHOT WOUND OF THE LEFT PHRENIC NERVE. Dr. F. H. Leavitt.

This man, a private in the army of Great Britain, aged 27 years, is shown as a case of possible gunshot wound of the left phrenic nerve. The history of the onset of the left chest disability, his symptoms and physical signs, roentgen-ray reports and the anatomic position of the pathway of the bullet are suggestive of left phrenic nerve paralysis. The confusing element is that, as a result of his gun shot wound, he has developed pleural adhesions in the left side of the chest, which interfere with normal respiratory movements; these are shown by fluoroscopic and roentgen-ray studies.

This patient received three perforating bullet wounds during his army service. The first occurred in 1915, in his left forearm. The next, in 1917, perforated his face. The third, in September, 1918, was by a bullet that entered at the left sternoclavicular junction, passed backward and came out at the spine of the scapula at the vertebral border. He was in the hospital for this injury only a comparatively short time. The anatomic position of the wound of entrance was about where the left phrenic nerve enters the mediastinum in close proximity to the subclavian artery and vein.

The patient complains principally of inspiratory dyspnea, especially on muscular exertion. There is marked limitation of movement of the entire left side of the chest and Littel's sign is absent on the left. He experiences difficulty in coughing and sneezing, but suffers no subjective sense of pain or other discomfort.

Roentgen-ray examination, made at the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases revealed: The fluoroscopic examination shows a normal diaphragm on the right side. On the left side, the diaphragm is raised and distorted in its outer third, with two lines running upward and outward that suggest adhesions between the pleura and the diaphragm. This is well shown on inspiration, when the diaphragm is pulled downward. It would seem that the adhesions are not sufficient to cause the marked distortion and collapse, and that some definite paralysis of the diaphragm must also be present. There is slight displacement of the heart to the right. On full expiration, the left side of the diaphragm assumes a more nearly normal shape, while, on full inspiration, the distortion produced by the apparent paralysis is much more marked.

I have never seen a case of gun shot paralysis of the phrenic nerve and this case impressed me as being one of that type; but, because of the confusion incident to the pleural adhesions, the diagnosis is not absolute.

THE SUCCESSFUL REMOVAL OF A SPINAL CORD GROWTH.

Dr. Tom A. Williams.

A woman, aged 39, was referred to me in January, 1923, because of a progressive loss of power in the legs and generally increasing, severe pains, which had begun in September, 1922. Pain had also occurred in the back and there was tingling in the lower part of the abdomen. She had had no acute illness at that time, but in the preceding June, a supposed uterine cancer had been removed by radium without a previous examination. The same surgeon reopened the abdomen in November because of intensity of pain; he had found nothing abnormal in the spine and nothing pathologic was discovered at the operation.

Examination revealed a woman slightly wasted, whose facies portrayed long suffering. She moved the left leg with difficulty but was unable to move the foot. There was marked atrophy of the left calf which measured 12¼ inches as compared with 13¾ inches on the right. The lower part of the left thigh was also much atrophied, the measurements being 15½ inches at the midway point and 19 inches on the right. The toes could be flexed, but dorsiflexion was practically absent. Both flexion and extension of the knee were much weaker than on the right side, where they were also diminished. The reflexes of the left leg were feeble; on the right, the patellar reflex was much exaggerated and there was ankle clonus, although the toes flexed on stroking the sole. The only disturbance of sensation was some indefinite hyperesthesia in the groins. There were no other neurologic signs. The spinal fluid was normal. The diagnosis was made of an extra medullary neoplasm implicating the third, fourth, and fifth lumbar roots on the left side and compressing the spinal cord at a level as high as the third lumbar segment. In view of the history this was, of course, believed to be a metastasis from the uterus.

Immediate operation was recommended as the best hope of a possible favorable prognosis. The operation which was performed by Dr. Horgan, revealed the dura mater bound down by a mass the size of a large bean at the level of the third lumbar segment. Neither spinal cord nor root was infiltrated and no xanthochromia appeared in the spinal fluid below. The growth was removed and proved to be, not a neoplasm, but a hemorrhagic pachymeningitis of chronic type. In part of the second lumbar vertebra, which appeared soft to the surgeon, no abnormality was found.

Since the operation, no severe pain has been felt by the patient and she has returned home comparatively comfortable. Complete recovery of motility awaits only time for regeneration of the anterior root fibers pressed on by the growth.

TRAUMA OF THE SPINE MISDIAGNOSED AS HYSTERIA.

Dr. Tom A. Williams.

The frequency of hysteria as the true interpretation of cases when compensation is in question should not be allowed to blind the physician to other possibilities. Mental inertia permits some men to fall into the error of what I have called diagnosis by categories. Traumatic hysteria was diagnosed by two psychiatrists in the case of a woman who was entitled to treatment by a compensation commission because of an injury to the neck. This was because, after a year's relief, intense pain again ensued with deviation and rigidity of

the neck. The pain and rigidity were not relieved by manipulation, although the patient had been completely relieved previously by manipulative treatment in the same hospital. By these psychiatrists the deviation was looked on as tic, and the whole situation was regarded as a psychic reaction to her former accident. A factor contributing to that diagnosis was the volubility of the patient and her excitable manner.

A more thorough exploration, however, made it clear that the patient's manner reflected, not lack of intellectual balance, but an earnestness of character aroused by a widow's necessity to gain a livelihood for her family. For twenty years she had proved herself highly efficient in responsible clerical work, in which she was eager to continue. Whatever anxiety she showed was attributed to the wish for recovery. All the movements of the head were restricted, more particularly bending to the right. The twist of the neck had none of the characters of mental torticollis. It was merely an attitude adopted to minimize pain.

Tics are convulsive and intemperate in character; are accompanied by consciousness of the act; are preceded by a desire sometimes mounting to a passion to perform the act; and are followed by a feeling of relief after performance of the movement. At all events, the victim of tics feels compelled to make the movement comprising the tic; hence the term impulsion or compulsion neurosis.

Although the roentgen-ray examination of the neck revealed no displacement of the vertebrae, there could be felt a distinct thickening, on the side of the spine towards which her head was turned, over the left transverse process of the third cervical vertebra. Accordingly I rejected the diagnosis of traumatic neurosis, believing that I was dealing with a trauma of the neck.

The treatment prescribed was manipulation by a skilled operator. The result was that the patient slept that night without the aid of the hypnotics she had been taking. Within a week she was completely free from pain and could move the neck in every direction. She has remained well for six months, although, as a precaution, manipulation was continued weekly.

PARALYSIS OF THE PERONEAL NERVE FROM OVERFLEXION OF THE LEG ON THE THIGH. Dr. William G. Spiller.

The peroneal nerve is frequently injured; recently an unusual cause of paralysis of this nerve has come under my observation. A young man was employed in laying hard wood floors and in his enthusiasm for doing the best that he could he over-exerted himself. He knelt on his left knee and kept the right leg flexed on the thigh at more than a right angle, with the sole of foot on the floor, and in this position reached as far forward as possible. In this way he produced severe pressure on the right peroneal nerve by the firmly contracted biceps muscle, the tendon of the muscle being close to the nerve. He developed a complete right sided peroneal palsy. Another man who had been long in the business stated that when he first began the work he also had a paralysis of the peroneal nerve.

CASE OF ACUTE EPIDEMIC ENCEPHALITIS. Dr. A. M. Ornsteen.

The occurrence of three characteristic symptom-types in rapid succession, with complete recovery in two weeks, are the points of especial interest in this case. The development of a state of acute generalized chorea, severe enough to be regarded as a case of acute chorea gravis, is also noteworthy.

The patient, a previously healthy woman, aged 27, became ill on Feb. 11, 1923, with grippal symptoms, for which she was treated during one week, being

partly confined to bed. On February 18, she suddenly experienced sharp pain in the left shoulder and left abdominal wall, associated with extreme hyperesthesia of the skin over these parts. The pains occurred at frequent intervals, were stabbing in character, and each pain was accompanied by gross jerking of the muscles in these areas. Forty-eight hours after the onset of these myoclonic pains, she suddenly became choreic, irritable and greatly disturbed emotionally. Within a few hours, every part of the head, body and limbs became involved in coarse, irregular jerkings, resembling, in every detail, Sydenham's chorea. Her mental condition was one of great irritability and anxiousness. This continued for forty-eight hours with marked insomnia and signs of beginning physical exhaustion. Great concern was felt about her condition at this time because of the similarity of the condition to the severe cases of acute chorea gravis. The above dyskinetic phenomena stopped as suddenly as they had begun with the appearance of lethargy and diplopia on the beginning of the third day. On the next day, partial bilateral ptosis was noted with slight turning inward of the left eye. The drowsiness and diplopia persisted for three days and then disappeared. Within two weeks from the onset of the severe pains, the patient had recovered completely from a symptomatic point of view. The only persistent symptom during the latter days was hyperesthesia of the left half of the abdomen and the left shoulder.

A rise in temperature was noted only when the chorea subsided and the lethargy appeared; it reached 101 F. by axilla, ran a remittent course for three days, and then only rose in the evening to 99.4 for three more days. A leukocytosis of 13,000 was present. General physical examination of heart and lungs was negative. A spinal fluid examination was not made, the clinical evidence being sufficient for the diagnosis.

ASPHYXIATION AT BIRTH CAUSED BY MENINGEAL HEMORRHAGE. Dr. W. B. Cadwalader.

I had a case recently of a child asphyxiated at birth because of difficulty in labor, although forceps had not been used. There were frequent severe general convulsions, as many as thirty in twenty-four hours; lumbar puncture showed bloody spinal fluid. The patient died after three weeks. At post-mortem examination a large subpial blood clot was found covering the anterior surface of the pons and medulla oblongata. My reason for speaking of this case is to emphasize the importance of asphyxiation at birth as a cause of meningeal hemorrhage.

SYPHILIS AS THE CAUSE OF MUSCULAR ATROPHY OF SPINAL ORIGIN. Drs. A. J. Ostheimer, Geo. Wilson, and N. W. Winkelman.

After a brief review of the literature in which the writings of Raymond, Léri, Dana and Spiller were especially referred to, the authors reported a number of cases of atrophy of spinal origin in which the evidence clearly pointed toward syphilis as the cause. One of these cases was with necropsy findings. The importance of treating all cases of muscular atrophy of spinal origin with antisyphilitic treatment was stressed. It was the opinion of the authors that this should be done despite negative blood and spinal fluid findings.

This paper will be published in full at a later date.

DISCUSSION

Dr. Charles S. Potts: I wish to add one case to this collection in which the patient was much improved by treatment. I saw this man about 1917. He worked in the Remington Arms Company at Chester where he had to use a hammer continuously. It is interesting that he first noticed weakness in his right arm with which he used the hammer. The atrophy was confined almost entirely to the shoulder girdles and muscles of the back, and there were fibrillary twitchings in these muscles. He had no pyramidal tract symptoms whatever. There was a positive Wassermann reaction; under the use of arsphenamin and mercury the process was arrested and he improved slightly. I saw the man within a year and there had been no progress whatever in his symptoms; if anything he was better.

Dr. J. Hendrie Lloyd: There is no doubt that syphilis can cause muscular atrophy. The wonder is that the spirochete does not invade the anterior horns and anterior roots more frequently than it seems to do. It is a well known fact that muscular atrophy occurs in advanced cases of tabes. I remember a number of such cases in our service at the Philadelphia Hospital; one case, in a woman, I put on record some 20 years ago. Some limbs looked as if she had no muscular tissue left. Nonne, in his monograph on syphilis, has presented pictures of patients with muscular atrophy due to syphilis. There is one phase of this subject to which I want to refer; that is the invasion of the lateral tracts. A few years ago Dr. Ludlum and I recorded cases of what we called primary lateral sclerosis in syphilitic patients. As is well known, lateral sclerosis occurs sometimes in association with muscular atrophy, as in amyotrophic lateral sclerosis. This raises the question whether amyotrophic lateral sclerosis may be due to syphilis.

Dr. A. J. Ostheimer: I want to stress the importance of the treatment. In view of the inefficacy of therapy of today in all forms of spinal muscular atrophy due to causes other than syphilis, the important point this paper is intended to bring out, is that all muscular atrophies should be treated as if they were due to syphilis. An improvement consequent on such treatment will be also a therapeutic aid in diagnosis. All these cases may be treated in the same way as other neurosyphilis, no matter whether it involves the lateral tracts, the posterior roots and columns, or other portions of the cord or brain.

INDEX TO VOLUME 9

The star (*) preceding the page number indicates an original article in THE ARCHIVES. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B,O and S, respectively.

PAGE

Aboulker, H.: Diagnosis of silent intracranial abscess in ambulatory stage 648
Abraham, K.: Manifestations of female castration complex 521
Actinomycosis, spinal; Lesné and Belloir 647
Agersborg, H. P. K.: Qualitative chemical and physical stimulations in nudibranchiate mollusks 377
Agnosia, transitory, of an infant in regard to its arms; Tournay 106
Agraphia, left hemiplegia with aphasia in left-handed person, and with agraphia in otherwise unaffected hand; Buckley 125
Alajouanine: Septum lucidum tumor 524
Alcoholic psychoses before and after prohibition; Pollock 378
psychosis associated with brain tumor; Burr 120
Amnesia, hysterical amnesia following physical injury; Read 533
Amyotonia and myopathy in same patient; Yudelson 669
Anatomy, experiments on development of cranial ganglia and lateral line sense organs in amblystoma punctatum; Stone 104
mutual relationship in development of brain and eyes of Lepidoptera; Kopéc 382
Angioma, venous, of cerebral cortex; Campbell and Ballance 235
of pons; Leyser 238
Apfelbach, C. W.: Traumatic fractures of cranial bones: edema of brain; bruises of brain 109
Apraxia, localization of; Forster 97
Argyll Robertson pupil; Wilson 242
Armstrong, H.: Retrobulbar neuritis with symptoms of spinal sclerosis 385
Arsphenamin therapy in state hospitals for mental disorders; G. W. Mills and C. L. Vaux *450
Arthritis, neuropathic; Duncan 383
Ascariasis, meningeal symptoms of; Turcan 525
Asphyxiation at birth caused by meningeal hemorrhage; Cadwalader 829
Asthenia and mania from antityphoid vaccination; Benon 800
Atrophy, Charcot-Marie; report of case with necropsy; C. S. Potts and G. Wilson *431
infantile progressive muscular atrophy of Werdnig-Hoffmann type; case report with necropsy; J. C. Michael *582
muscular, of spinal origin; syphilis as cause of; Ostheimer, Wilson and Winkelman 829
muscular, progressive; Courtney 113
Austria, neurology and psychiatry in; Blitzsten 273
Automatism, ambulatory; H. D. Singer *347
Autosuggestion: See Suggestion
Avelis syndrome; Gordon 121
Ayer, J. B.: Subtentorial cyst of meninges 403

Babinski sign, is there a Babinski sign of the hand? Radovici 386
Bacillus acidophilus, implantation of, in persons with psychoses; L. A. Julianelle and F. G. Ebaugh *769

PAGE

Bagby, E.: Etiology of phobias 523
Bailey, P.: Experimental diabetes insipidus 102
Recent developments in electrodiagnosis *436
Unusual tumor of third ventricle in diabetes insipidus 807
Baldi, E.: Physiology of nervous system of insects; research on circus movements provoked in coleoptera 234
Baldie, A.: Hysteria 383
Ballance, C.: Venous angioma of cerebral cortex 235
Barbital intoxication; Stearns, 529; Sands 819
treatment of insane epileptics; I. A. Darling *478
Barlow, R. A.: Vestibular nerve function in myxedema 525
Bassoe, P., and Davis, C. B.: Two cases of brain tumor with ventriculography *178, 263
Bausch: Elementary convulsions in catatonia 95
Belloir: Spinal actinomycosis 647
Benon, R.: Asthenia and mania from antityphoid vaccination 800
Berkheiser, E. J.: Neuropathic arthropathies—Charcot's spine 385
Bernheim: Pain in gangrene of extremities 234
Bertrand: Septum lucidum tumor 524
Bessière, R.: Thorium X in psychiatric therapeutics 230
Bickel, G.: Recovery from tuberculous meningitis 108
Syndrome of the central gray nuclei 218
Blastomycosis, cystic, of cerebral gray matter; caused by torula histolytica Stoddard and Cutler; W. Freeman and F. D. Weidman *589
Blitzsten, N. L.: Neurology and psychiatry in Austria and Germany 273
Blood creatinin findings in 5 cases of corpus striatum disorder; Raphael and Potter 651
studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito 792
sugar in blood and spinal fluid; Polonovski and Duhot 646
Body and mind: origin of dualism; Mott 381
Bolton, G. C.: Hysterical gangrene 642
Book Reviews:
Anomale Kinder; L. Scholz 133
Brain Abscess; Its Surgical Pathology and Operative Technic; W. P. Eagleton 415
Concerning Psychanalysis; introductory presentation by Allers; edited by Stransky and Dattner 673
Constitutional Factors in Dementia Praecox with Particular Attention to the Circulatory System and to Some of the Endocrine Glands; N. D. C. Lewis 686
Die Epidemische Encephalitis; F. Stern 684
Elements of Scientific Psychology; K. Dunlap 132
Functional Nervous Disorders: Their Classification and Treatment; D. E. Core 282
Kingdom of Evils; Psychiatric Social Work Presented in 100 Case Histories, Together with a Classification of Social Divisions of Evil; E. E. Southard, M. C. Jarrett and R. C. Cabot 545
Mental Diseases. A Public Health Problem; J. V. May 281
Multiple Sclerosis (Disseminated Sclerosis) 685
Nervous Ills, Their Cause and Cure; B. Sidis 544
Outwitting Our Nerves; J. A. Jackson and H. M. Salisbury 416
Psychic Health of Jesus; W. E. Bundy 135
Syphilis of the Innocent; H. C. Solomon and M. H. Solomon 134
Botulism, epidemic of; Morel and de St. Martin 651

PAGE

Boultier: Benzoin colloidal reaction 644
Bowman, K. M.: Mental disorder with low basal metabolism 259
—and Grabfield, G. P.: Basal metabolism in mental disease *358
Boyd, T. E.: Studies on visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals 96
Brain abscess, diagnosis of silent intracranial abscess in ambulatory stage; Aboulker 648
abscess, traumatic, of anterior cerebral fossa; Courtney 113
calcification, unusual; Weimann 107
edema, studies in traumatic fractures of cranial bones: edema of brain; bruises of brain; Apfelbach 109
fundamental plan of vertebrate brain; Kingsbury 108
hemorrhage in young adults; 2 cases; Burley 803
mutual relationship in development of brain and eyes of Lepidoptera; Kopéc 382
points of view concerning forebrain morphology in lower vertebrates; Holmgren 111
spirochetes in syphilitic endarteritis of brain; Sioli 787
stabilizing brain tissue during fixation; G. S. Stevenson *763
syndrome of central gray nuclei; Bickel 218
syphilis and psychoses; Redalié 790
tumor, acute alcoholic psychosis associated with brain tumor; Burr 120
tumor, cases illustrating some aspects of brain and spinal cord tumors; Elsberg 388
tumor, endothelioma of brain 1 year after operation; Freeman 540
tumor, enchondroma of brain removed with recovery; Spiller 536
tumor, glioma of cerebral hemispheres; 2 cases; Throckmorton 384
tumor, mixed cell sarcoma of; Crafts 381
tumor of septum lucidum; Souques, Alajouanine and Bertrand 524
tumor, presentation of case in which presumptive diagnosis of tumor had been made 3 years before; Frazier 538
tumor, 2 cases with ventriculography; P. Bassoe and C. B. Davis *178, 263
tumor, unusual case of brain tumor; Watermann 527
tumor, unusual tumor of third ventricle in case of diabetes insipidus; Bailey and Reifenstein 807
tumor, venous angioma of cerebral cortex; Campbell and Ballance 235
Bremer, F.: Experimental diabetes insipidus 102
Bresowsky: Lesions of subthalamic region 101
Broadwin, J. T.: Spinal spastic paralysis 237
Buck, C. F.: Social service work in neurological clinic at the Massachusetts General Hospital during 1922 406
Buckley, A. C.: Left hemiplegia with aphasia in left-handed person, and with agraphia in the otherwise unaffected hand 125
Burgess, H.: Basal metabolism in nontoxic goiter and borderline thyroid cases 99
Burley, B. T.: Cerebral hemorrhage in young adults 803
Burr, C. W.: Acute alcoholic psychosis associated with brain tumor 120
Buscaino, V. M.: Dysthyroidism, anaphylaxis, and epilepsy 801
Buzzard, E. F.: Traumatic and toxic ulnar neuritis 382
Byrnes, C. M.: Singultus crisis in tabes dorsalis 279
Byrnes, M. A.: Study of spinal accessory nerves from a case of bilateral acquired spasmodic torticollis 130

Cadwalader, W. B.: Apoplectiform onset of left temporal lobe symptoms probably due to encephalitis 822
Asphyxiation at birth caused by meningeal hemorrhage 829
Herpes zoster and tabes dorsalis 119
Paralysis of upward associated movements of eyeballs 278
Campbell, H.: Venous angioma of cerebral cortex 235
Cannon, W. B.: Experimental traumatic shock; the critical level in falling blood pressure 91

PAGE

Capps, J. A.: Localization of pain sense in parietal and diaphragmatic peritoneum 645
Carbon monoxide (illuminating gas) poisoning: report of case presenting unusual sequela; Wilson 665
Carlson, A. J.: Studies on visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals 96
Carniol: Oculogastric and oculocolic reflexes 227
Castration complex, manifestations of female castration complex; Abraham 521
Catatonia, elementary convulsions in; Bausch 95
schizophrenic, with associated metabolic and vegetative features, report of case; T. Raphael, J. P. Parsons and M. N. Woodwell *471
Catola, G.: Parkinson's disease following encephalitis 514
Cattell, M.: Experimental traumatic shock; the critical level in falling blood pressure 91
Central gray nuclei, syndrome of; Bickel 218
Cerebellum, atrophy in adults, with report of a clinical case; Arrigo Frigerio 372
demonstration of animals in which results of recent lesions of cerebellar cortex of one side were contrasted with those resulting from extirpation of otic labyrinth of the same side; Pike 247
genesis of cerebellar functions; F. Tilney *137, 246
hemorrhage, case of congenital megacolon (Hirschsprung's disease) associated with mental disorder and terminal cerebellar hemorrhage; Lewis 519
tumor: operation: relief of symptoms; Taylor 396
Cerebrospinal fluid, benzoin colloidal reaction in 105 neurologic cases; Marie, Boultier and Iorgoulesco 644
changes following air injection; Hermann 384
reaction of colloidal benzoin in general paralysis; Targowla 644
source, distribution and circulation; Stewart 243
studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito 792
sugar in blood and spinal fluid; Polonovski and Duhot 646
viscosity of; Soda 650
Cestan: Diagnostic value of spinal fluid Wassermann 111
Chadman, M. G.: Myasthenia gravis of slow development 116
Charcot joint of elbow in Jewish woman; Lindauer 410
Charcot's spine; Ridlon and Berkheiser 385
Charcot-Marie atrophy: report of case with necropsy; C. S. Potts and G. Wilson *431
Charles, J. R.: Manganese poisoning 244
Cheney, C. O.: Focal infection: its presence and elimination in functional psychoses 392
Cheney, G. W. H.: Colloidal gold reaction in acute poliomyelitis 241
Children, actual environment of children in a Boston district; Taylor 260
Chordotomy, section of anterolateral columns of spinal cord (chordotomy); physiologic effects and clinical results in 8 cases; C. H. Frazier and W. G. Spiller *1
Chorea, clinicopathologic study of acute and chronic chorea; G. Wilson and N. W. Winkelman *170
hemichorea of sudden onset in which there were no other symptoms; Gilpin 664
hemiplegia with hemichorea; Remond and Colombies 103
Sydenham's, psychoses associated with; Hammes 387
Clark, L. P.: Epileptoid or fainting attacks in hypopituitarism 239
Importance of special educational training for mental defect dependent upon organic lesions 373
Clymer, G.: Chronic lead poisoning vs. tabes dorsalis 397
Hyoscin in Parkinsonian rigidity 399

PAGE

Cole, W. H.: Transplantation of skin in frog tadpoles 94
Colella, R.: Association between tuberculosis, neuropsychopathy and delinquency 640
Coleman, G. H.: Localization of pain sense in parietal and diaphragmatic peritoneum 645
Colombies, H.: Hemiplegia with hemichorea 103
Colon, congenital megacolon (Hirschsprung's disease) associated with mental disorder and terminal cerebellar hemorrhage; Lewis 519
Contracture, similar hysterical contractures in wife and husband; Weisenburg 824
Convulsions, clinical picture and prognosis of frequent minor fits of children; Stier 520
experimental; a consideration of epileptogenous zones of central nervous system; L. J. Pollock *604
studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man; preliminary report; C. A. Elsberg and B. P. Stookey *613
Corpus callosum, results of puncture of; Pohlisch 99
Corpus striatum disorder, blood creatinin findings in 5 cases; Raphael and Potter 651
Cotton, H. A.: Functional psychoses 223
Courtney, J. W.: Progressive muscular atrophy 113
Traumatic abscess of anterior cerebral fossa 113
Crafts, L. M.: Mixed cell sarcoma of brain 381
Cramer, A.: Recovery from tuberculous meningitis 108
Cranium, studies in traumatic fractures of cranial bones: edema of brain; bruises of brain; Apfelbach 109
Cycloplegia in girl of 12; Stearns 532

Daday, P.: Thorium X in psychiatric therapeutics 230
Da Fano, C.: Changes of Golgi's apparatus in nerve cells of spinal cord following exposure to cold 111
Dana, C. L.: Ecology of epilepsy; epilepsy in colonies *551
Danielopolu: Oculogastric and oculocolic reflexes 227
Danysz, J.: Origin of psychic energy 362
Darling, I. A.: Phenobarbital (luminal) treatment of insane epileptics *478
Davenport, C. B.: Ecology of epilepsy; racial and geographic distribution of epilepsy *554
Davis, C. B., and Bassoe, P.: Two cases of brain tumor with ventriculography *178, 263
Davis, L. E.: Deep sensibility of face *283
Davis, T. K.: Double papilledema 245
Decerebrate rigidity in animals and its recognition in man; Walshe 112
on the production of neuro-muscular patterns by release of spinal integrations after decerebration; Kraus and Rabiner 239
Delgado, H. F.: Treatment of paresis by inoculation with malaria 522
Delinquency, association between tuberculosis, neuropsychopathy and delinquency; Rosolino Colella 640
Dementia praecox, blood analyses in catatonic dementia praecox; Uyematsu and Soda 112
genetic origin of dementia praecox; Mott 380
severe changes in nuclei of ganglion cells in dementia praecox; Stocker 519
sex development and behavior in male patients with dementia praecox; C. E. Gibbs *73
Depression, case with feelings of unreality; Morris 258
Detwiler, S. R.: Studies on retina: photomechanical responses in retina of eremias argus 800

PAGE

Devine, H.: An expiation process in schizophrenia 233
Diabetes insipidus, experimental; Bailey and Bremer 102
Diaphragm center in cerebral cortex and hiccup; Knapp 90
Doyle, J. B.: Glossopharyngeal neuralgia *34
Dualism, origin of; Mott 381
Duhot, E.: Sugar in blood and spinal fluid 646
Duncan, J. H.: Neuropathic arthritis 383
Dunham, H. G.: After-care of infantile paralysis cases of 1916 epidemic in Brooklyn 796
Dunn, A. D.: Pachymeningitis hemorrhagica interna 386
Dyspituitarism: See Pituitary
Dysthyroidism: See under Thyroid
Dystonia lenticularis; Solomon, 256; Frigerio 365
Dystrophy, pathologic findings in heart muscle in progressive muscular dystrophy; J. H. Globus *59
progressive muscular dystrophy; Friedman 236

Ebaugh, F. G., and Julianelle, L. A.: Implantation of bacillus acidophilus in persons with psychoses *769
Education, importance of special educational training for mental defect dependent upon organic lesions; Clark 373
Electrodiagnosis, recent developments in; P. Bailey *436
Elsberg, C. A.: Sensory disturbances in tumors of spinal cord and membranes 656
Brain and spinal cord tumors 388
—and Stookey, B. P.: Studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man *613
Embolism, occipital lobe embolism; Woods 648
Encephalitis, acute epidemic encephalitis; Ornsteen 828
apoplectiform onset of left temporal lobe symptoms probably due to encephalitis; Cadwalader 822
clinical considerations on Parkinson's disease following encephalitis; Catola 514
clinical notes on pathology of epidemic encephalitis complicated by a psychosis; G. B. Hassin and D. B. Rotman *22
epidemic, pathologic study of 5 cases including 2 with myoclonia; M. E. Morse *751
lesions in brain of patient with postencephalitic paralysis agitans; J. C. McKinley *47
lethargica; Hall 371
pathological findings in chronic progressive encephalitis; Marinesco 371
sequels, spasmodic forced respiration as sequel of epidemic encephalitis; Pardee 800
sequels, 4 cases; Ornsteen 667
sequels, review of clinical sequelae of recent epidemic of lethargic encephalitis; Naville 515
status of epidemic encephalitis as an independent disease 633
treatment of residual epidemic encephalitis; Hyslop 387
twenty cases of lethargic encephalitis with pathological findings in 4 cases; Hume, Nattrass and Shaw 370
Enchondroma of brain removed with recovery; Spiller 536
Endarteritis, spirochetes in syphilitic endarteritis of brain; Sioli 787
Endocrines, some endocrine aspects of the psyche (lecture delivered to Upsala Medical Society); Hammar 368
Endothelioma of brain 1 year after operation; Freeman 540
Enfield, C. D.: Normal sella 387
Epilepsy and bilious attacks; Vining 243
and the convulsive state; F. Kennedy *567
ecology of epilepsy; epilepsy in colonies; C. L. Dana *551

PAGE

Epilepsy—Continued
ecology of epilepsy; racial and geographic distribution of epilepsy; C. B. Davenport *554
experimental convulsions; a consideration of epileptogenous zones of central nervous system; L. J. Pollock *604
focal, 3 cases; Mixter 402
mechanism and fundamental cause of epilepsies; J. Rosett *689
pathogenesis of epilepsy; critical review, with new interpretation of available data; M. Osnato 254, *488
phenobarbital (luminal treatment of insane epileptics; I. A. Darling .. *478
some problems in; W. Timme *547
studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man; preliminary report; C. A. Elsberg and B. P. Stookey *613
symptomatic epilepsy in birds; an anatomico-pathologic and clinical report; G. Mingazzini *576
Epileptoid or fainting attacks in hypopituitarism; Clark 239
Esophagus, visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals; Carlson, Boyd and Pearcy 96
Eyman, E. V.: Case with unusual mental symptoms 662

Face, deep sensibility of; L. E. Davis *283
Fay, T.: Administration of hypertonic salt solutions for relief of intracranial pressure 539
—and Grant, F. C.: Lesions of optic chiasm and tracts with relation to adjacent vascular structures *739
Feeblemindedness, hereditary and nonhereditary mental defect, a comparative study; H. W. Potter and R. S. Viers *339
Flatau, E.: Cervical neurofibroma 524
"Nuquo-mydriasis" sign 523
Forster: Localization of apraxia 97
Foster, N. B.: Metabolic states contributing to uremia *627
Frazier, C. H.: Presentation of case in which presumptive diagnosis of tumor had been made 3 years before 538
—and Spiller, W. G.: Section of anterolateral columns of spinal cord (chordotomy) *1
Freeman, W.: Endothelioma of brain 540
Glioma of medulla 541
—and Weidman, F. D.: Cystic blastomycosis of cerebral gray matter *589
Friedman, E. D.: Progressive muscular dystrophy 236
Frigerio, A.: Atrophy of cerebellum in adults 372
Dystonia lenticularis progressiva 365
Froin's syndrome and its relation to allied conditions in spinal fluid; Greenfield 241

Gangrene, hysterical; Bolton 642
pain in threatened and real gangrene of extremities; Bernheim 234
Gaté: Antityphoid vaccination and generalized scleroderma 802
Gerard, M. W.: Afferent impulses of trigeminal nerve; intramedullary course of painful, thermal and tactile impulses *306
Germany, impressions of neurology in Russia and Germany; Hassin 268
Gibbs, C. E.: Sex development and behavior in male patients with dementia praecox *73
Gilpin, S. F.: Erb's type of spinal syphilis 664
Hemichorea of sudden onset 664
Glia reactions, concerning diffuse glia reactions; Pines 236
Glioma of medulla; Freeman 541
Globus, J. H.: Histopathology of amaurotic family idiocy 816
Pathologic findings in heart muscle in progressive muscular dystrophy ... *59
Teratoid cyst of hypophysis *417

PAGE

Glosso-pharyngeal neuralgia, 4 cases; J. B. Doyle *34
Goetsch test of hyperthyroidism 635
Goiter, basal metabolism in nontoxic goiter and in borderline thyroid cases, with reference to its bearing in differential diagnosis; Means and Burgess 99
exophthalmic, therapy of 781
Goldsmith, W. M.: Process of ingestion in the ciliate, Frontonia 231
Golf, excessive golf followed by musculospiral palsy; Wholey 386
Gordon, A.: Avelis syndrome 121
Mother and daughter presenting symptoms of degenerative state of central nervous system 667
Paramyotonia 276
Sensory disturbance limited to portion of distribution of ulnar nerve 411
Grabfield, G. P., and Bowman, K. M.: Basal metabolism in mental disease ..*358
Graff: Pituitrin as muscular tonic 107
Grant, F. C.: Peripheral nerve tumor 542
—and Fay, T.: Lesions of optic chiasm and tracts with relation to adjacent vascular structures *739
Grant, S. B.: Tetany 100
Greenfield, J. G.: Froin's syndrome 241
Gregg, D.: Physiology of autosuggestion 115

Hall, A. J.: Encephalitis lethargica 371
Hallucination, experimental study of mechanism of; Prince 232
Hammar, J. A.: Endocrine aspects of the psyche 368
Hammes, E. M.: Psychoses with Sydenham's chorea 387
Hassin, G. B.: Neurology in Russia and Germany 268
Solitary tubercle of spinal cord 228
—and Rotman, D. B.: Epidemic encephalitis complicated by a psychosis ... *22
Haviland, C. F.: Mental hygiene 369
Hayaschi, Y.: Antiproteolytic ferments, amylase and oxydase in psychosis 637
Hemiplegia, left, with aphasia in left-handed person, and with agraphia in the otherwise unaffected hand; Buckley 125
syphilitic, with unusual trophic disturbances; Patten 275
with hemichorea; Remond and Colombies 103
Henderson, D. K.: War psychoses 244
Hermann, G.: Spinal fluid following air injection 384
Herpes, association of herpes zoster and tabes dorsalis; Cadwalader 119
Hiccup, diaphragm center in cerebral cortex and hiccup; Knapp 90
singultus crisis in tabes dorsalis; Byrnes 279
Holmes, W. H.: Chronic trophedema (Milroy-Meige disease) 266
Holmgren, N.: Forebrain morphology in lower vertebrates 111
Hospitals, need of psychopathic departments in state hospitals; Ross 378
psychiatric institute-hospital; Pilcher 375
Hoyt, R.: Value of neurological examination in syphilis 801
Hume, W. E.: Lethargic encephalitis 370
Hyperthyroidism: See under Thyroid
Hypophysis: See Pituitary
Hyslop, G. H.: Residual epidemic encephalitis 387
Hysteria; Baldie 383
hysterical amnesia following physical injury; Read 533
hysterical gangrene; Bolton 642
similar hysterical contractures in wife and husband; Weisenburg 824

Idiocy, amaurotic family, histopathology of; Globus 816

PAGE

Immunology, studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to the immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito 792
Infection, focal, is the stomach a focus of infection? Kopeloff..... 649
focal; its presence and elimination in functional psychoses; Kopeloff and Cheney 392
Intracranial pressure, administration of hypertonic salt solutions for relief of; Fay 539
Iorgoulesco: Benzoin colloidal reaction..... 644

Jacobi, W.: General protein therapy and active immunization of general paralysis 517
Jacoby, J. R.: Memorial to William M. Leszynsky..... 817
Jahnel, F.: Spirochetes in central nervous system in general paralysis..... 511
Jaloustre, L.: Thorium X in psychiatric therapeutics..... 230
Jelliffe, S. E.: Modern school of Dutch neuropsychiatry..... 658
Julianelle, L. A., and Ebaugh, F. G.: Implantation of bacillus acidophilus in persons with psychoses *769

Keidel, A.: Asymptomatic neurosyphilis; apparent rôle of immunity in genesis of neurosyphilis..... 240
Kennedy, F.: Epilepsy and the convulsive state.....*567
Extrathecal abscess of spinal cord..... 652
Vertebral dislocation 653
Kingsbury, B. F.: Fundamental plan of vertebrate brain..... 108
Knapp: Diaphragm center in cerebral cortex and hiccup..... 90
Kopéc, S.: Influence of nervous system on development and regeneration of muscles and integument in insects..... 797
Mutual relationship in development of brain and eyes of Lepidoptera.... 382
Kopeloff, N.: Focal infection: its presence and elimination in functional psychoses 392
Is stomach a focus of infection?..... 649
Krabbe, K. H.: Tuberose sclerosis and hydrocephalus..... 105
Kraus, W. M.: Difference between muscular and neuromuscular interpretation of walking *184
Phylogeny and classification of spinal motor integrations..... 812
Production of neuro-muscular patterns..... 239
Kuhlmann tests; psychiatric importance of; Plant..... 530
Kuntz, A.: Reflex arcs in myenteric and submucous plexuses..... 377

Lead poisoning, chronic, versus tabes dorsalis; Clymer..... 397
Leavitt, F. H.: Gunshot wound of left phrenic nerve..... 826
Juvenile general paralysis with onset at 7 years..... 825
Legrand, R.: Aortitis and trigeminal neuralgia..... 526
Neo-arsphenamin in hypodermic injections..... 229
Lennox, W. G.: Neurosyphilis among Chinese..... *26
Lesné: Spinal actinomycosis 647
Leszynsky, William M. Leszynsky, memorial to; Jacoby..... 817
Lewis, N. D. C.: Congenital megacolon (Hirschsprung's disease) with mental disorder 519
Leyser: Angioma of the pons..... 238
Morphine poisoning 651
Lindauer, E.: Charcot joint of elbow..... 410
Lipodystrophy, case of; Patten 408
Little's Disease: See Paralysis, spastic
Locomotor Ataxia: See Tabes
Lorente y Patrón, S.: Treatment of general paralysis with relapsing fever virus 650

PAGE

McDonald, C. A.: Examination of heart and aortic arch by roentgen rays.. 406
McFarland, A. R.: Involvement of nervous system in various phases of syphilis 798
McKinlay, C. A.: Effect of extract of posterior lobe of pituitary on basal metabolism 110
McKinley, J. C.: Lesions in brain of patient with postencephalitic paralysis agitans *47
Manganese poisoning; 3 cases; Charles..... 244
Mania and asthenia from antityphoid vaccination; Benon..... 800
Marie, P.: Benzoin colloidal reaction..... 644
Marinesco, M. G.: Chronic progressive encephalitis..... 371
Means, J. H.: Basal metabolism in nontoxic goiter and in borderline thyroid cases 99
Medulla, glioma of; report of case with exhibition of specimen; Freeman... 541
Mejía Xesspe, T.: Peruvian folk lore..... 646
Meninges, hemorrhage, asphyxiation at birth caused by; Cadwalader..... 829
meningeal symptoms of ascariasis; Turcan..... 525
subtentorial cyst of meninges, with necropsy; Ayer..... 403
Meningioma, both intradural and extradural, removed from spinal cord with complete recovery; Spiller 534
Meningitis, tuberculous, recovery from; Cramer and Bickel..... 108
Mental defect, hereditary and nonhereditary mental defect, a comparative study; H. W. Potter and R. S. Viers..... *339
hygiene, actual environment of children in a Boston district; Taylor..... 260
hygiene, facts and fancies of; Pratt..... 808
hygiene needs arising subsequent to school life; Haviland..... 369
symptoms, case with unusual mental symptoms; Eyman..... 662
Metabolism, basal, effect of extract of posterior lobe of pituitary on basal metabolism in normal individuals and in those with endocrine disturbances; McKinlay 110
basal, in mental disease; K. M. Bowman and G. P. Grabfield..... *358
basal, in nontoxic goiter and in borderline thyroid cases, with particular reference to its bearing in differential diagnosis; Means and Burgess 99
basal, presentation of 5 cases of mental disorder with low metabolism; Bowman 259
metabolic states contributing to uremia; N. B. Foster..... *627
nature of certain functional nervous disturbances and their treatment along metabolic lines; R. Pemberton..... *208, 280
study of antiproteolytic ferments, amylase and oxydase in 4 types of psychosis, with especial consideration of metabolism in mental disease; Hayaschi 637
Michael, J. C.: Infantile progressive muscular atrophy of Werdnig-Hoffmann type *582
Microscopy, stabilizing brain tissue during fixation; G. S. Stevenson..... *763
Mills, G. W., and Vaux, C. L.: Arsphenamin therapy in state hospitals for mental disorders *450
Minet, J.: Aortitis and trigeminal neuralgia..... 526
Neo-arsphenamin in hypodermic injection..... 229
Mingazzini, G.: Symptomatic epilepsy in birds..... *576
Minnich, D. E.: Tarsal sensitivity to solutions of saccharose, in red admiral butterfly, Pyrameis atalanta Linn..... 379
Mixter, W. J.: Focal epilepsy..... 402
Monoplegia, brachial, due to thrombosis of subclavian vein; Wilson..... 383
Moore, J. E.: Asymptomatic neurosyphilis; influence of pregnancy on incidence of neurosyphilis 649
Morel, C.: Epidemic of botulism..... 651
Morphin poisoning, unusual case of; Leyser..... 651
Morris, C. H.: Depression with feelings of unreality..... 258

PAGE

Morse, M. E.: Epidemic encephalitis *751

Motor activity, psychopathologic and localizing value of parallelism between psychic and motor activity; Steck 798

Mott, F. W.: Body and mind: origin of dualism 381

Genetic origin of dementia praecox 380

Movement, difference between muscular and neuromuscular interpretation of walking; W. M. Kraus *184

practical bearing on diagnosis of phylogeny and classification of spinal motor integrations; Kraus 812

Muscles, influence of nervous system on development and regeneration of muscles and integument in insects; Kopec 797

Myasthenia gravis of slow development; Spiller and Chadman 116

Myoclonias; Roger 797

Myopathy and amyotonia in same patient; Yudelson 669

Myxedema, vestibular nerve function in; Barlow 525

Naito, K.: Spinal fluid and blood of syphilitic and normal persons 792

Nathan, P. W.: Lumbar arthritis as cause of sciatica 818

Nattrass, F. J.: Lethargic encephalitis 370

Recurrent hypertrophic neuritis 238

Naville, F.: Sequelae of lethargic encephalitis 515

Neo-arsphenamin in hypodermic injections; Minet and Legrand 229

Nerve, abducens, researches on abducens nerve and particularly on significance of its accessory nucleus; Terni 783

artificial nerve branches for innervation of paralyzed muscles; Stookey .. 249

facial, deep sensibility of the face; L. E. Davis *283

phrenic, gunshot wound of left phrenic nerve; Leavitt 826

trigeminal, afferent impulses of trigeminal nerve; intramedullary course of painful, thermal and tactile impulses; M. W. Gerard *306

trigeminal, deep sensibility of the face; L. E. Davis *283

tumor, 2 cases of peripheral nerve tumor; Grant 542

ulnar, case of sensory disturbance limited to portion of distribution of ulnar nerve; Gordon 411

Nervous system, influence of, on development and regeneration of muscles and integument in insects; Kopec 797

mother and daughter presenting symptoms of a degenerative state of central nervous system; Gordon 667

nature of certain functional nervous disturbances and their treatment along metabolic lines; R. Pemberton *208, 280

physiology of nervous system of insects; research on circus movements provoked in coleoptera; Baldi 234

sympathetic, afferent paths of sympathetic system, with special reference to nerve cells of spinal ganglia sending their peripheral processes into the rami communications; Rossi 382

syphilis, comparative clinical observations on involvement of nervous system in various phases of syphilis; Stokes and McFarland 798

syphilis, Erb's type of spinal syphilis presenting difficulties in diagnosis; Gilpin 664

syphilis, examination of heart and aortic arch by roentgen rays in neurosyphilis; McDonald 406

syphilis, neurorecurrences following treatment with arsphenamin; Zimmermann 106

syphilis, neurosyphilis among Chinese; W. G. Lennox *26

syphilis, studies in asymptomatic neurosyphilis; apparent influence of pregnancy on incidence of neurosyphilis in women; Moore 649

syphilis, studies in asymptomatic neourosyphilis: apparent rôle of immunity in genesis of neurosyphilis; Keidel 240

syphilis, value of neurological examination in; Hoyt 801

vibrating sensation in diseases of nervous system; Williamson 793

visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals; Carlson, Boyd and Pearcy 96

PAGE

Neuralgia, glosso-pharyngeal, 4 cases; J. B. Doyle..... *34
trigeminal, and aortitis; Minet and Legrand..... 526
Neuritis, recurrent, hypertrophic; Nattrass..... 238
retrobulbar, associated with symptoms and signs of disseminated spinal sclerosis; Armstrong 385
ulnar, some varieties of traumatic and toxic ulnar neuritis; Buzzard..... 382
Neurofibroma, cervical; Flatau and Sawicki..... 524
Neurology in Austria and Germany; Blitzsten..... 273
Neuropsychiatry, reflections on the modern school of Dutch neuropsychiatry; Jelliffe 658
Neuropsychopathy, association between tuberculosis, neuropsychopathy and delinquency; Rosolino Colella 640
Neurosyphilis: See Nervous System, syphilis
Nicholas: Antityphoid vaccination and generalized scleroderma..... 802
Nixon, C. E.: Spinal fluid and blood of syphilitic and normal persons..... 792
"Nuquo-mydriasis" sign; Flatau 523
Nystagmus, significance of nystagmus in a lesion of abducens nucleus; Rosett 232

OBITUARIES:
Edward Mercur William, M.D., 1881-1823..... 778
Robert Thaxter Edes 506
Occupational therapy as means of controlling disturbed patients; Ryon.... 385
Optic chiasm and tracts, lesions of, with relation to adjacent vascular structures; T. Fay and F. C. Grant.....*739
Ornsteen, A. M.: Acute epidemic encephalitis..... 828
Postencephalitic sequelae..... 667
Osnato, M.: Pathogenesis of epilepsy.....254, *488
Ostheimer, A. J.: Syphilis as cause of muscular atrophy of spinal origin... 829

Pachymeningitis hemorrhagica interna: study of 5 cases of nontraumatic hemorrhagic spinal fluid; Dunn..... 386
Pain, localization of pain sense in parietal and diaphragmatic peritoneum; Capps and Coleman 645
Palsy: See Paralysis
Papilledema, double, caused by blocking of cord at fourth cervical vertebra, greatly relieved by operation; Davis..... 245
Paralysis agitans in girl of 12; Stearns..... 531
agitans, clinical considerations on Parkinson's disease following encephalitis; Catola 514
agitans, hyoscin in Parkinsonian rigidity; Clymer..... 399
agitans, lesions in brain of patient with postencephalitic paralysis agitans; J. C. McKinley *47
artificial nerve branches for innervation of paralyzed muscles; Stookey. 249
Avelis syndrome; Gordon 121
Brown-Sequard paralysis from dislocation of cervical vertebra; Viets.... 399
excessive golf followed by musculospiral palsy; Wholey..... 386
familial infantile bulbar palsy; Paulian..... 526
general protein therapy and active immunization of general paralysis; Jacobi 517
general, reaction of colloidal benzoin in general paralysis; Targowla.... 644
general, spirochetes in central nervous system in general paralysis; Jahnel 511
general, treatment with relapsing fever virus; Lorente y Patrón..... 650
general, treatment by inoculation with malaria; Wagner-Jauregg, 522; Delgado 522
juvenile general paralysis with onset at 7 years; Leavitt..... 825
of peroneal nerve from overflexion of leg on thigh; Spiller..... 828
of upward associated movements of eyeballs; Cadwalader..... 278
spastic, some ocular phases of Little's disease; Posey..... 802
spinal spastic paralysis (primary lateral sclerosis); J. T. Broadwin..... 237

PAGE

Paramyotonia, case of; Gordon 276
Pardee, I. H.: Spasmodic forced respiration as sequel of encephalitis 800
Paresis: See Paralysis, general
Parker, G. H.: Crawling of young loggerhead turtles toward sea 243
Parkinson's disease: See Paralysis agitans
Parsons, J. P., Woodwell, M. N., and Raphael, T.: Schizophrenic catatonia with associated metabolic and vegetative features *471
Patten, C. A.: Lipodystrophy 408
Syphilitic hemiplegia 275
Paulian, E. D.: Familial infantile bulbar palsy 526
Pearcy, J. F.: Viceral sensory nervous system; reflex control of cardia and lower esophagus in mammals 96
Pellagra, two cases; Smith 257
Pemberton, R.: Functional nervous disturbances and their treatment along metabolic lines *208, 280
Peruvian folk lore; Mejia Xesspe 646
Pfahler: Calcification of pituitary with hypopituitarism and with symptomatic treatment 240
Pfister, O.: Plato: a forerunner of psychoanalysis 525
Phobia, etiology of; Bagby 523
Physiology, crawling of young loggerhead turtles toward the sea; Parker ... 243
process of ingestion in the ciliate, frontonia; Goldsmith 231
Pick's visions; Stenvers 794
Pike, F. H.: Animals in which the results of recent lesions of cerebellar cortex of one side were contrasted with those resulting from extirpation of the otic labyrinth of same side 247
Pilcher, L. F.: Psychiatric institute-hospital 375
Pines, J. L.: Diffuse glia reactions 236
Pitfield: Calcification of pituitary with hypopituitarism and with symptomatic treatment 240
Pituitary, calcification of pituitary with hypopituitarism and with symptomatic treatment; Pfahler and Pitfield 240
epileptoid or fainting attacks in hypopituitarism; Clark 239
extract, effect of extract of posterior lobe of pituitary on basal metabolism in normal individuals and in those with endocrine disturbances; McKinlay 110
extract, further facts regarding influence of feeding anterior lobe of hypophysis on rate of growth and size of ambystoma tigrinum; Uhlenhuth 802
teratoid cyst of hypophysis; J. H. Globus *417
Pituitrin as muscular tonic; an ergographic study; Urechia and Graff 107
Plant, J. S.: Psychiatric importance of Kuhlmann tests 530
Pohlisch: Results of puncture of corpus callosum 99
Poliomyelitis, after-care of infantile paralysis cases of 1916 epidemic in Brooklyn; Dunham 796
value of routine use of colloidal gold reaction in acute epidemic poliomyelitis; Regan and Cheney 241
Pollock, H. M.: Alcoholic psychoses before and after prohibition 378
Pollock, L. J.: Experimental convulsions; consideration of the epileptogenous zones of central nervous system *604
Polonovski: Sugar in blood and spinal fluid 646
Pons, angioma of; Leyser 238
tubercle of tegmentum pontis—contribution to symptomatology of pontine lesions—Pick's visions; Stenvers 794
Popper, E.: Cause of schizophrenia 104
Development of schizophrenia 93
Posey, W. C.: Ocular phases of Little's disease 802

PAGE

Potter, F. C.: Blood creatinin findings in corpus striatum disorder..... 651
Potter, H. W., and Viers, R. S.: Hereditary and nonhereditary mental defect *339
Potts, C. S., and Wilson, G.: Charcot-Marie atrophy.....*431
Pratt, G. V.: Facts and fancies of mental hygiene..... 808
Prince, M.: Experimental study of mechanism of hallucinations..... 232
Psychanalysis. Plato: a forerunner of psychanalysis; Pfister..... 525
Psychiatry, histopathologic studies in; Spielmeyer..... 639
Psychic activity, psychopathologic and localizing value of parallelism between psychic and motor activity; Steck..... 798
energy, origin of; Danysz..... 362
Psychology in medicine; Wells..... 376
Psychopathic inferiority, constitutional; Visher..... 375
Psychosis, alcoholic, before and after prohibition; Pollock..... 378
alcoholic, associated with brain tumor; Burr..... 120
and cerebral syphilis; Redalié..... 790
associated with Sydenham's chorea; Hammes..... 387
basal metabolism in mental disease; K. M. Bowman and G. P. Grabfield..*358
clinical and pathologic differentiation of senile psychoses and arteriosclerotic psychoses; Vonderahe 122
etiology and treatment of the so-called functional psychoses; summary of results based upon experience of 4 years; Cotton..... 223
implantation of bacillus acidophilus in persons with psychoses; L. A. Julianelle and F. G. Ebaugh.....*769
preliminary study of the precipitating situation in 200 cases of mental disease; Strecker 102
presentation of 5 cases of mental disorder with low basal metabolism; Bowman 259
studies in focal infection; its presence and elimination in functional psychoses; Kopeloff and Cheney..... 392
study of antiproteolytic ferments, amylase and oxydase in 4 types of psychosis, with special consideration of metabolism in mental disease; Hayaschi 637
war; the infective exhaustive group; Henderson..... 244
Puberty, precocious, tuberose sclerosis and hydrocephalus and their association with precocious puberty; Krabbe..... 105
Pulawski, A.: Thymic death..... 799
Pupil, Argyll Robertson; Wilson 242
Pyramidal tracts, injury of one pyramidal tract resulting from knitting needle puncture wound of cervical region of cord; Yaskin..... 413

Rabiner, A. M.: Production of neuro-muscular patterns..... 239
Radovici, A.: Is there a Babinski sign of hand?..... 386
Oculogastric and oculocolic reflexes..... 227
Palm-chin reflex 98
Raphael, T.: Blood creatinin findings in corpus striatum disorder..... 651
—Parsons, J. P., and Woodwell, M. N.: Schizophrenic catatonia with associated metabolic and vegetative features.....*471
Raynor, M. W.: Social work in state hospital..... 242
Read, C. F.: Hysterical amnesia following physical injury..... 533
Ream, M. J.: Temperament in harmonious human relationship..... 524
Redalié, L.: Cerebral syphilis and psychoses..... 790
Reflex arcs, occurrence of, in myenteric and submucous plexuses; Kuntz.... 377
oculogastric and oculocolic reflexes; Danielopolu, Radovici and Carniol.. 227
palm-chin reflex; Radovici 98
Regan: Colloidal gold reaction in acute poliomyelitis..... 241
Reifenstein, B. J.: Unusual tumor of third ventricle in diabetes insipidus... 807
Remond, A.: Hemiplegia with hemichorea..... 103

PAGE

Retina, studies of retina; photomechanical responses in retina of eremias argus; Detwiler 800
Ridlon, J.: Neuropathic arthropathies—Charcot's spine..... 385
Riser: Diagnostic value of spinal fluid Wassermann..... 111
Roger, H.: The myoclonias..... 797
Rosett, J.: Mechanism and fundamental cause of epilepsies.....*689
Significance of nystagmus in a lesion of abducens nucleus..... 232
Ross, J. R.: Need of psychopathic department in state hospitals..... 378
Rossi, O.: Afferent paths of sympathetic nervous system..... 382
Rotman, D. B., and Hassin, G. B.: Pathology in case of epidemic encephalitis complicated by a psychosis..... *22
Russia, impressions of neurology in; Hassin..... 268
Ryon, W. G.: Occupational therapy to control disturbed patients..... 385

Saccharose, quantitative study of tarsal sensitivity of solutions of saccharose, in red admiral butterfly, Pyrameis atalanta Linn; Minnich 379
de St. Martin, R.: Epidemic of botulism..... 651
Sands, I. J.: Barbital intoxication..... 819
Sawicki, B.: Cervical neurofibroma 524
Schizophrenia, and expiation process in case of schizophrenia; Devine.... 233
clinical studies on cause of; Popper..... 104
clinical studies on development of; Popper..... 93
schizophrenic catatonia with associated metabolic and vegetative features; T. Raphael, J. P. Parsons and M. N. Woodwell.....*471
Sciatica, lumbar arthritis as cause of; Nathan..... 818
Scleroderma, antityphoid vaccination and generalized scleroderma; Nicholas and Gaté 802
Sclerosis, combined, of spinal cord of anemic type without blood changes; Winkelman 412
multiple, spirochete stain in multiple sclerosis; G. S. Stevenson..... *88
Sella turcica, normal sella; Enfield..... 387
Sergent's cutaneous white line; Sézary..... 110
Sézary: Cutaneous white line (of Sergent)..... 110
Shaw, A. F. B.: Lethargic encephalitis..... 370
Shock, experimental traumatic shock; the critical level in falling blood pressure; Cannon and Cattell..... 91
Singer, H. D.: Ambulatory automatism.....*347
Anosmia and agensia..... 262
Sioli, F.: Spirochetes in syphilitic endarteritis of brain..... 787
Skin, transplantation of skin in frog tadpoles, with reference to adjustment of grafts over the eyes, and to local specificity of integument; Cole... 94
Skull: See Cranium
Smell, an unusual syndrome; anosmia and agensia; Singer..... 262
Smith, S. K.: Pellagra 257
Social service, organization of social work in state hospital; Raynor..... 242
resumé of social service work in neurological clinic at Massachusetts General Hospital during 1922; Buck..... 406

Society Transactions:
Boston Society of Psychiatry and Neurology.....113, 256, 396, 527, 803
Chicago Neurological Society 262, 531, 669
New York Neurological Society.....245, 388, 652, 812, 817
New York Neurological Society and Neurological Section of the Academy of Medicine 248
Philadelphia Neurological Society.....116, 275, 408, 534, 662, 822
Soda, T.: Blood analyses in catatonic dementia praecox..... 112
Viscosity of cerebrospinal fluid..... 650

PAGE

Solomon, H. C.: Dystonia lenticularis 256
Souques: Septum lucidum tumor 524
Spielmeyer, W.: Histopathologic studies in psychiatry 639
Spiller, W. G.: Enchondroma of brain removed with recovery 536
Meningioma, both intradural and extradural, removed from spinal cord .. 534
Myasthenia gravis 116
Paralysis of peroneal nerve from overflexion of leg on thigh 828
—and Frazier, C. H.: Section of anterolateral columns of spinal cord (chordotomy) *1
Spinal cord, abscess, case of extrathecal abscess of cord; Taylor and Kennedy 652
changes of Golgi's apparatus in nerve cells of spinal cord following exposure to cold; Da Fano 111
section of anterolateral columns of spinal cord (chordotomy); discussion of physiologic effects and clinical results in series of 8 cases; C. H. Frazier and W. G. Spiller *1
sensory disturbances in tumors of spinal cord and membranes, and their diagnostic significance; Elsberg 656
tubercle, clinico pathological notes on solitary tubercle of spinal cord; Thalhimer and Hassin 228
tumor, meningioma both intradural and extradural, removed from spinal cord with complete recovery; Spiller 534
tumor, successful removal of spinal cord growth; Williams 827
tumors, cases illustrating some aspects of brain and spinal cord tumors; Elsberg 388
Spine, case of vertebral dislocation; reduction; Taylor and Kennedy 653
trauma of spine misdiagnosed as hysteria; Williams 827
Spirochetes in syphiltic endarteritis of brain; Sioli 787
Stearns, A. W.: Barbital intoxication 529
Sterns, W. G.: Cycloplegia in girl of 12 532
Paralysis agitans in girl of 12 531
Steck, H.: Psychopathologic and localizing value of parallelism between psychic and motor activity 798
Stenvers, H. W.: Tubercle of tegmentum pontis 794
Stevenson, G. S.: Spirochete stain in multiple sclerosis *88
Stabilizing brain tissue during fixation *763
Stevenson, L.: Comparative study of icebox and water bath fixation methods in Wassermann as applied to nervous diseases 248
Stewart, R. M.: Spinal fluid; source, distribution, and circulation 243
Stier, E.: Prognosis of frequent minor fits of children 520
Stocker, A.: Severe changes in nuclei of ganglion cells in dementia praecox 519
Stoddart, W. H. B.: Mental factor in visceroptosis 387
Stokes, J. H.: Involvement of nervous system in various phases of syphilis. 798
Stomach, is the stomach a focus of infection? Kopeloff 649
Stone, L. S.: Experiments on development of cranial ganglia and lateral line sense organs in amblystoma punctatum 104
Stookey, B.: Artificial nerve branches for innervation of paralyzed muscles 249
—and Elsberg, C. A.: Studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man *613
Strecker, E. A.: The precipitating situation in 200 cases of mental disease .. 102
Suggestion, physiology of autosuggestion; Gregg 115
Syphilis: See also Nervous system, syphilis of
arsphenamin therapy in state hospitals for mental disorders; G. W. Mills and C. L. Vaux *450
cerebral, and psychoses; Redalié 790
studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito 792

PAGE

Tabes dorsalis, association of herpes zoster and tabes dorsalis; Cadwalader 119
chronic lead poisoning versus tabes dorsalis; Clymer 397
singultus crisis in tabes dorsalis; Byrnes 279
Targowla, R.: Reaction of colloidal benzoin in general paralysis 644
Taste, an unusual syndrome: anosmia and agensia; Singer 262
Taylor, A. S.: Extrathecal abscess of spinal cord 652
Vertebral dislocation; reduction 653
Taylor, E. W.: Cerebellar tumor: operation: relief of symptoms 396
Taylor, M.: Actual environment of children in a Boston district 260
Temperament in harmonious human relationship; Ream 524
Teratoid cyst of the hypophysis; J. H. Globus *417
Termopsis, castes of; Thompson 109
Terni, T.: Researches on the abducens nerve 783
Tetany; report of cases with acid-base disturbance; Grant 100
Thalamus, contribution to our knowledge of lesions of subthalamic region; Bresowsky 101
Thalhimer, W.: Solitary tubercle of spinal cord 228
Thompson, C. B.: Castes of Termopsis 109
Thorium X in psychiatric therapeutics; Daday, Bessière and Jaloustre .. 230
Throckmorton, T. B.: Glioma of cerebral hemispheres 384
Thymus, new contribution to study of thymic death; Pulawski 799
Thyroid, dysthyroidism, anaphylaxis and epilepsy; Buscaino 801
hyperthyroidism, test of 635
Tilney, F.: Genesis of cerebellar functions *137, 246
Timme, W.: Epilepsy *547
Torticollis, study of spinal accessory nerves from case of bilateral acquired spasmodic torticollis; Byrnes 130
Tournay, A.: Transitory agnosia of infant 106
Trophedema, chronic trophedema (Milroy-Meige disease); report of 2 cases; Holmes 266
Tuberculosis, association between tuberculosis, neuropsychopathy and delinquency; Rosolino Colella 640
tubercle of tegmentum pontis—contribution to symptomatology of pontine lesions—Pick's visions; Stenvers 794
Tumor: See Brain tumor; Pons, tumor; Spinal cord tumor, etc.
Turcan, H.: Meningeal symptoms of ascariasis 525
Typhoid vaccination and generalized scleroderma; Nicholas and Gaté 802
vaccination, asthenia and mania from; Benon 800

Uhlenhuth, E.: Influence of feeding anterior lobe of hypophysis on rate of growth and size of ambystoma tigrinum 802
Urechia: Pituitrin as muscular tonic 107
Uremia, metabolic states contributing to; N. B. Foster *627
Uyematsu, S.: Blood analyses in catatonic dementia praecox 112

Vaux, C. L., and Mills, G. W.: Arsphenamin therapy in state hospitals for mental disorders *450
Ventriculography, spinal fluid changes following air injection; Hermann ... 384
two cases of brain tumor with; P. Bassoe and C. B. Davis *178, 263
Vertigo and its treatment; Woolsey 400
Vibrating sensation in diseases of nervous system; Williamson 793
Viers, R. S., and Potter, H. W.: Hereditary and nonhereditary mental defect *339
Viets, H. R.: Brown-Sequard paralysis from dislocation of cervical vertebra 399
Vining, C. W.: Bilious attacks and epilepsy 243

PAGE

Visceroptosis, mental factor in visceroptosis; Stoddart 387
Visher, J. W.: Constitutional psychopathic inferiority 375
Vonderahe, A. R.: Differentiation of senile psychoses and arteriosclerotic psychoses 122

Wagner-Jauregg: Treatment of general paresis by inoculation of malaria .. 522
Walking, difference between muscular and neuromuscular interpretation of walking; W. M. Kraus *184
Walshe, F. M. R.: Decerebrate rigidity 112
Wassermann reaction, comparative study of icebox and water bath fixation methods in Wassermann reaction as applied to nervous diseases; Stevenson 248
diagnostic value of spinal fluid Wassermann reaction; Cestan and Riser .. 111
Watermann, G. A.: Brain tumor 527
Weidman, F. D., and Freeman, W.: Cystic blastomycosis of cerebral gray matter *589
Weimann: Calcification in brain 107
Weisenburg, T. H.: Similar hysterical contractures in wife and husband .. 824
Wells, F. L.: Psychology in medicine 376
White line of Sergent (cutaneous white line); Sézary 110
Wholey, C. C.: Excessive golf followed by musculospiral palsy 386
Williams, T. A.: Successful removal of spinal cord growth 827
Trauma of spine misdiagnosed as hysteria 827
Williamson, R. T.: Vibrating sensation in diseases of nervous system 793
Wilson, G.: Brachial monoplegia due to thrombosis of subclavian vein 383
Illuminating gas poisoning 665
Syphilis as cause of muscular atrophy of spinal origin 829
—and Potts, C. S.: Charcot-Marie atrophy *431
—and Winkelman, N. W.: Acute and chronic chorea *170
Wilson, S. A. K.: Argyll Robertson pupil 242
Winkelman, N. W.: Combined sclerosis of spinal cord of anemic type without blood changes 412
Syphilis as cause of muscular atrophy of spinal origin 829
—and Wilson, G.: Acute and chronic chorea *170
Woods, A. H.: Occipital lobe embolism 648
Woodwell, M. N., Raphael, T., and Parsons, J. P.: Schizophrenic catatonia with associated metabolic and vegetative features *471
Woolsey, C. L.: Vertigo and its treatment 400

Yaskin, J. C.: Injury of one pyramidal tract resulting from knitting needle puncture wound of cervical region of cord 413
Yudelson, A. B.: Myopathy and amyotonia in same patient 669

Zimmermann, E. L.: Neurorecurrences following arsphenamin 106

THE ARCHIVES OF NEUROLOGY AND PSYCHIATRY is published by the American Medical Association to stimulate research in the field of diseases and disorders of the nervous system, and to disseminate knowledge in this department of medicine.

Manuscripts for publication, books for review, and correspondence relating to the editorial management should be sent to Dr. T. H. Weisenburg, Editor-in-Chief, 1909 Chestnut St., Philadelphia, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF NEUROLOGY AND PSYCHIATRY, American Medical Association, 535 North Dearborn Street, Chicago.

Articles are accepted for publication on condition that they are contributed solely to the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. Manuscripts must be typewritten, preferably double spaced, and the original copy submitted. Zinc etchings and halftones of illustrations will be supplied by the publisher when the original illustrations warrant.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index published by the American Medical Association. This requires, in the order given: name of author, title of article, name of periodical, with volume, page, month—day of month if weekly—and year. On receipt of stamped addressed envelope, "Bibliographic Style," containing rules for footnotes and bibliographies, and a list of leading medical periodicals of the world, with approved abbreviations, will be sent. "Suggestions to Medical Authors," a comprehensive pamphlet on the preparation of medical manuscripts, which also includes "Bibliographic Style," will be sent on receipt of twenty-five cents.

Authors will receive one hundred reprints free; additional reprints may be obtained at cost.

The domestic subscription price per year of two volumes is $6.00; Canadian, $6.40; Foreign, $6.75, including postage. Single copies, 75 cents, postpaid.

Make checks, etc., payable to the AMERICAN MEDICAL ASSOCIATION.

CONTENTS

PAGE

THE MECHANISM AND THE FUNDAMENTAL CAUSE OF THE EPILEPSIES. JOSHUA ROSETT, M.D., NEW YORK 689

LESIONS OF THE OPTIC CHIASM AND TRACTS WITH RELATION TO THE ADJACENT VASCULAR STRUCTURES. TEMPLE FAY, M.D., AND FRANCIS C. GRANT, M.D., PHILADELPHIA 739

EPIDEMIC ENCEPHALITIS. A PATHOLOGIC STUDY OF FIVE CASES INCLUDING TWO WITH MYOCLONIA. M. E. MORSE, M.D., BOSTON 751

STABILIZING BRAIN TISSUES DURING FIXATION. GEORGE S. STEVENSON, M.D., VINELAND, N. J. 763

IMPLANTATION OF BACILLUS ACIDOPHILUS IN PERSONS WITH PSYCHOSES. L. A. JULIANELLE, M.D., AND F. G. EBAUGH, M.D., PHILADELPHIA 769

OBITUARY: EDWARD MERCUR WILLIAMS, M.D. 778

NEWS AND COMMENT:

THE THERAPY OF EXOPHTHALMIC GOITER 781

ABSTRACTS FROM CURRENT LITERATURE 783

THE ABDUCENS NERVE AND ITS ACCESSORY NUCLEUS.—SPIROCHETES IN SYPHILITIC ENDARTERITIS OF THE BRAIN.—CEREBRAL SYPHILIS AND PSYCHOSES.—THE CEREBROSPINAL FLUID AND BLOOD OF SYPHILITIC AND NORMAL PERSONS.—THE VIBRATING SENSATION IN NERVOUS DISEASES.—TUBERCLE OF THE TEGMENTUM PONTIS.—AFTER-CARE OF INFANTILE PARALYSIS.—THE MYOCLONIAS.—DEVELOPMENT AND REGENERATION OF MUSCLES AND INTEGUMENT IN INSECTS.—THE PSYCHOLOGIC AND LOCALIZING VALUE OF PARALLELISM BETWEEN PSYCHIC AND MOTOR ACTIVITY.—COMPARATIVE CLINICAL OBSERVATIONS ON THE INVOLVEMENT OF THE NERVOUS SYSTEM IN SYPHILIS.—THYMIC DEATH.—SPASMODIC FORCED RESPIRATION AS A SEQUEL OF EPIDEMIC ENCEPHALITIS.—PHOTOCHEMICAL RESPONSES OF THE RETINA OF EREMIAS ARGUS.—ASTHENIA AND MANIA FROM ANTITYPHOID VACCINATION.—NEUROLOGIC EXAMINATIONS IN SYPHILIS.—DYSTHYROIDISM, ANAPHYLAXIS AND EPILEPSY.—THE INFLUENCE OF FEEDING ANTERIOR LOBE OF HYPOPHYSIS ON THE RATE OF GROWTH AND SIZE OF AMBYSTOMA TIGRINUM.—SOME OCULAR PHASES OF LITTLE'S DISEASE.—ANTITYPHOID VACCINATION AND GENERALIZED SCLERODERMA.

SOCIETY TRANSACTIONS:

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY 803

NEW YORK NEUROLOGICAL SOCIETY 812

PHILADELPHIA NEUROLOGICAL SOCIETY 822

INDEX TO VOLUME 831